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Angiographic Diagnosis of Expanding Intracranial Lesions by Vascular Displacement[1]

CARL F. LIST, M.D., and FRED J. HODGES, M.D.

Ann Arbor, Mich.

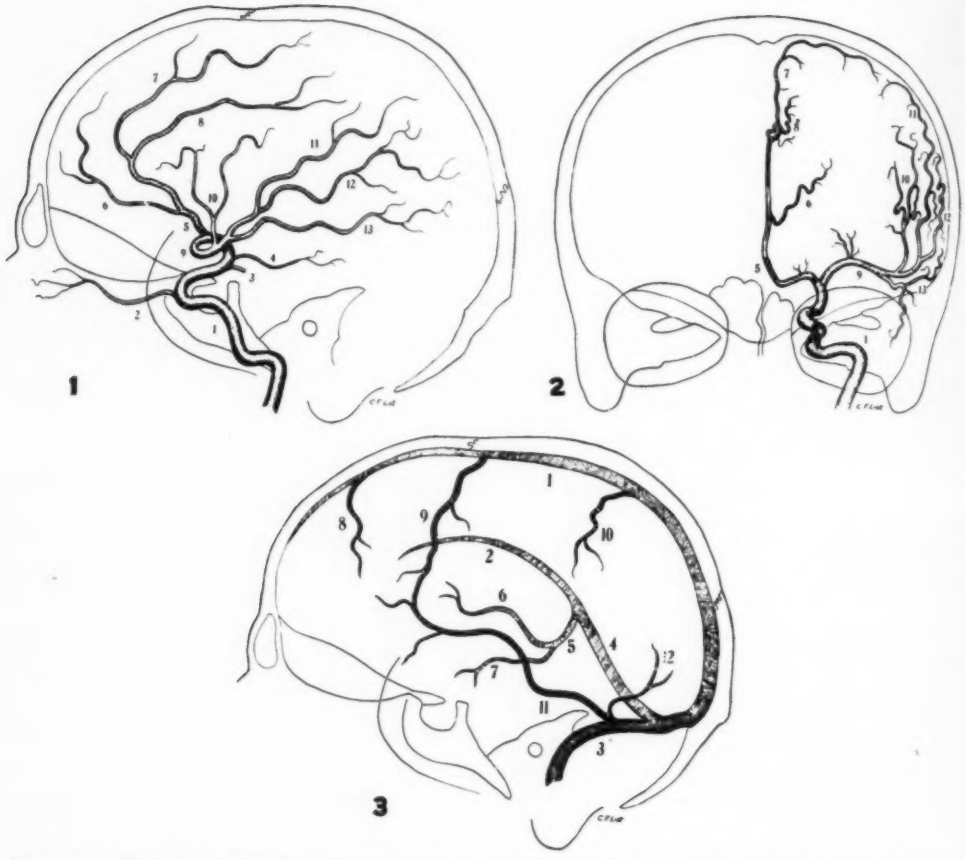
THROUGH THEIR combined efforts, neurosurgeons and roentgenologists have accomplished much to place the diagnosis of expanding intracranial lesions upon an exact basis. Pneumoencephalography, and particularly ventriculography, have been highly successful in the analysis of obscure situations. The more recently developed procedure of intracranial angiography is another standard method of neurosurgical diagnosis. To a certain extent angiography and ventriculography are interchangeable; more importantly, they supplement each other, each having its advantages and limitations. In cases of brain tumor, angiography has the following advantages:

1. It is a safe procedure, even in the presence of high intracranial pressure, as it avoids traumatizing punctures of the diseased brain and does not materially interfere with intracranial hydrodynamics; the patients, therefore, face major surgical intervention in better condition following angiography than after ventriculography. This is especially true of comatose subjects in whom, as a rule, roentgenographic contrast methods are indispensable for an accurate diagnosis. In such cases ventriculography is notoriously dangerous and may jeopardize the patient's dwindling chances of survival, whereas angiography may provide as much or even more information at a far lesser risk. A total of 194 angiographies, among which were 136 cases of expanding intracranial lesion, were performed in the University Hospital without a single death attributable to the procedure. Serious immediate complications, such as cerebral thrombosis or late radioactive damage due to the use of thorotrast, were not observed.

2. Angiography may give information both as to the location and the anatomic type of the suspected lesion. The site of the lesion is revealed by localized displacement of cerebral blood vessels. The angiographic picture is most striking if the tumor encroaches on large vessels, *i.e.*, if it is located not far from the surface of the brain. Conversely, tumors buried deeply in the cerebral substance or within the ventricular cavities are less clearly demonstrated. The anatomic type of the lesion can be frequently recognized by a characteristic manner of vascular deformation and by a specific pathologic vascular pattern within the lesion itself. Moreover, in cases with doubtful diagnosis, angiography not only rules out tumor (as does ventriculography), but may contribute positive evidence for a vascular lesion.

From the above considerations the following indications for angiography have been evolved:

[1] From the Department of Surgery, Section of Neurosurgery, and the Department of Roentgenology, University of Michigan Medical School, Ann Arbor, Mich. Accepted for publication in March 1946.



Figs. 1–3. Schematic drawings of normal arteriograms and venogram obtained by injection of the internal carotid artery.

Figs. 1 and 2. Arteriograms in lateral and anteroposterior projections.

1. Internal carotid artery. 2. Ophthalmic artery. 3. Posterior communicating artery. 4. Anterior choroidal artery. 5. Anterior cerebral artery. 6. Frontopolar artery. 7. Callosomarginal artery. 8. Pericallosal artery. 9. Middle cerebral artery. 10. Ascending frontoparietal artery. 11. Posterior parietal artery. 12. Angular artery. 13. Posterior temporal artery.

Fig. 3. Venogram in lateral projection.

1. Superior sagittal sinus. 2. Inferior sagittal sinus. 3. Transverse sinus. 4. Straight sinus. 5. Great cerebral vein of Galen. 6. Internal cerebral vein. 7. Basal vein of Rosenthal. 8. Frontal ascending vein. 9. Rolandic vein of Trolard. 10. Parietal ascending vein. 11. Communicating temporal vein of Labbé. 12. Descending temporo-occipital vein.

1. In certain expanding lesions of the base of the brain and skull.

2. In space-occupying lesions of the cerebral hemisphere which have been grossly localized, or at least lateralized, by other diagnostic methods.

3. In the differential diagnosis of neoplasm *versus* vascular lesion (especially aneurysm or vascular malformation).

On the other hand, angiography is definitely inferior to ventriculography:

1. In cases of increased intracranial pressure without any localization.

2. In tumors of the ventricular system, basilar ganglia, midbrain, and posterior fossa.

For the angiographic technic, the reader is referred to an earlier paper (1). It may be briefly mentioned here that satisfactory diagnostic information is secured from a

stereoscopic set of arteriograms and venograms in lateral projection and a single arteriogram in anteroposterior projection (total of 5 films). Diagrammatic drawings of normal carotid arteriograms in lateral and anteroposterior projections and of a venogram in lateral projection are presented here to facilitate the understanding of the pathologic pictures to follow (Figs. 1, 2, and 3).

This study deals with the characteristic displacement of cerebral vessels caused by space-occupying lesions. In a subsequent publication the specific vascular pattern within intracranial neoplasms will be discussed. The present report is based on angiographic examination of 136 patients with expanding intracranial lesions[2] treated at the University of Michigan Hospital from 1941 to 1946. The material will be grouped in the following manner:

1. General angiographic aspects of expanding intracranial lesions.
2. Tumors of the basilar surface of the brain.
3. Tumors of the temporal lobe.
4. Tumors of the occipital lobe.
5. Tumors of the frontal lobe.
6. Tumors of the parietal lobe.
7. Tumors of the basal ganglia and thalamus.

1. GENERAL ANGIOGRAPHIC ASPECTS OF EXPANDING INTRACRANIAL LESIONS

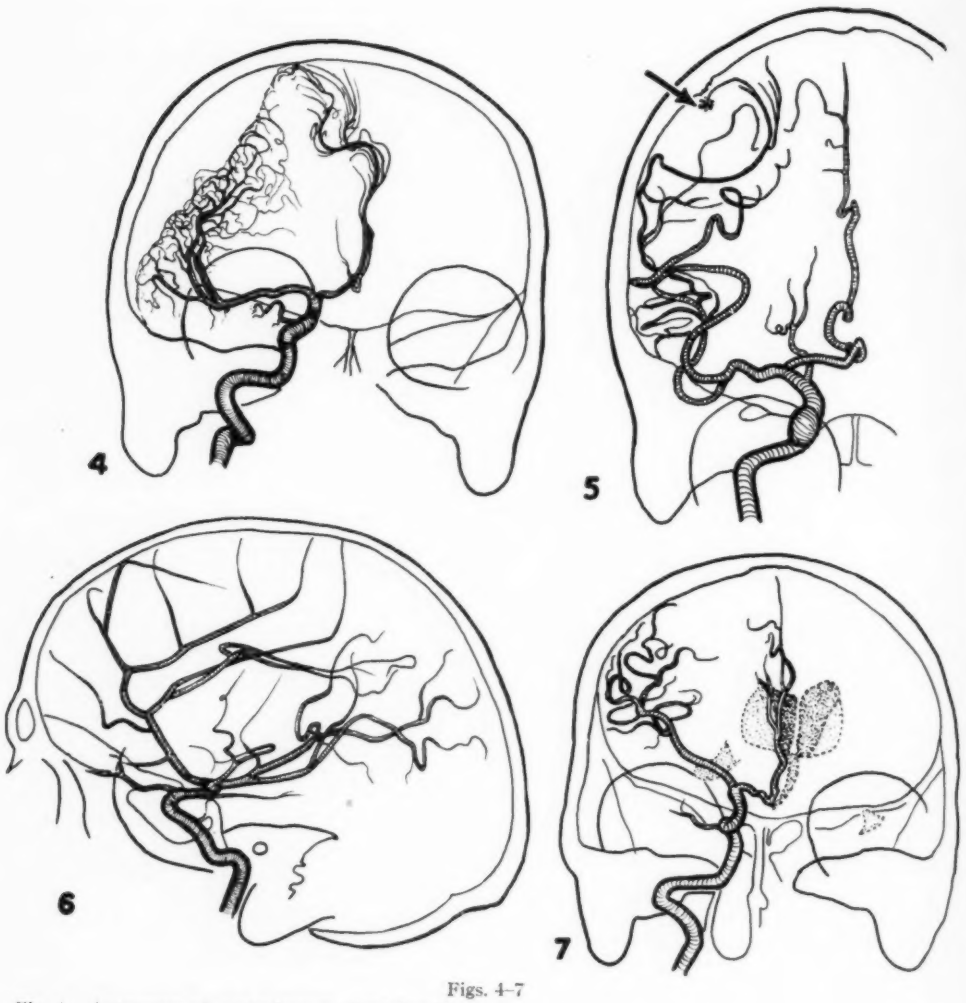
Expanding intracranial lesions produce either localized or generalized enlargement of the brain volume by (*a*) their own bulk or the presence of cysts and hemorrhages, (*b*) accompanying brain swelling, (*c*) enlargement of the ventricles. These changes result in typical deformities of intracranial blood vessels. Normally the arteries of the brain take a remarkably tortuous course, frequently forming complete loops. Any localized space-occupying process tends to displace the vessels away from the lesion. Thus, the arteries become separated from one another, thinned, and stretched, and they follow a straightened course. The cerebral veins, too, are displaced, but usually engorged. In cases of brain tumor, visualization of cerebral veins frequently is better than in cases with normal intracranial pressure, because of venous stasis and general slowing of cerebral blood circulation. Since the cerebral veins have a variable arrangement, their deformation and displacement are of only limited diagnostic value.

The type of vascular displacement depends upon three variable factors, *viz.*, the location, size, and pathologic type of the lesion. In epicerebral expanding lesions, such as epidural or subdural hematoma, the cortical vessels are displaced and separated from the convexity of the skull by an interposed area devoid of vessels. This is characteristically demonstrated only on the anteroposterior film, which gives an "optic cross section" of the lesion (Fig. 4).[3] In circumscribed superficial tumors (*e.g.*, meningioma), an area of spread and displaced vessels is surrounded by a corona of crowded (but otherwise normal) blood vessels. Here again the anteroposterior view may more graphically portray the extent of the lesion in depth than the lateral projection (Figs. 5 and 38). If a tumor is diffusely infiltrating, deeply located, and accompanied by considerable swelling of the surrounding brain, the vessels, especially the arteries, are spread and stretched over a wide area (Fig. 6).

Any sizable enlargement of a cerebral hemisphere causes shifting of the midline structures such as the anterior cerebral artery. Normally, this vessel keeps in the midline or transgresses it over short stretches only if it happens to possess great physiologic tortuousness. The changed position of the anterior cerebral artery often is not quite so sensitive an indicator of an expanding hemispheral process as is the midline shift of the ventricular system (Fig. 7). Obvious displacement of the anterior cerebral artery *per se* does not reveal a specific localiza-

[2] Among them 122 true neoplasms.

[3] Since satisfactory reproduction of original roentgenograms proved difficult, one of us (C.F.L.) made line drawings by careful tracing of the films.

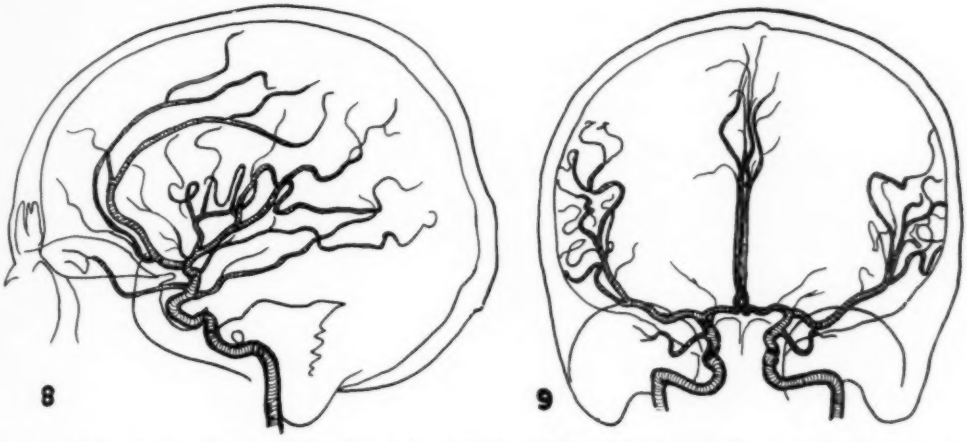


Figs. 4–7

Fig. 4. Anteroposterior arteriogram of subdural hematoma.
Fig. 5. Anteroposterior arteriogram of parietal meningioma. Arrow points to special circulation at attachment of tumor.
Fig. 6. Lateral arteriogram of diffuse astrocytoma of frontal and parietal lobes.
Fig. 7. Simultaneous arteriogram and ventriculogram in anteroposterior projection, meningioma of the middle fossa.

tion of the space-occupying lesion. Since the rigid falx resists the pressure of an enlarged hemisphere, the upper medial edge of the hemisphere is less displaced than the lower and anterior parts in the midline plane, which tend to shift underneath the falx. This is shown by a definite notching of the anterior cerebral artery around the lower edge of the falx (Figs. 4 and 21).

Ventricular enlargement cannot be recognized in angiograms with the same precision as in ventriculograms; in fact, it can be diagnosed only by indirect evidence. Internal hydrocephalus produces elevation and rounding out of the anterior half of the anterior cerebral artery *without* midline shift (Figs. 8 and 9) (2, 3). This is explained by the elevation of the corpus callosum, the contour of which is marked by the pericallosal artery. In extreme hy-



Figs. 8 and 9. Arteriograms of obstructive internal hydrocephalus, lateral projection and simultaneous bilateral arteriogram in anteroposterior projection.

drocephalus, also, the arteries of the convexity appear to be stretched, with the sylvian vessels taking a straight diagonal course. Other less reliable signs for severe hydrocephalus are, according to Egas Moniz (4), elevation of the inferior sagittal sinus, elongation of the straight sinus, depression of the internal cerebral vein and of the great vein of Galen.

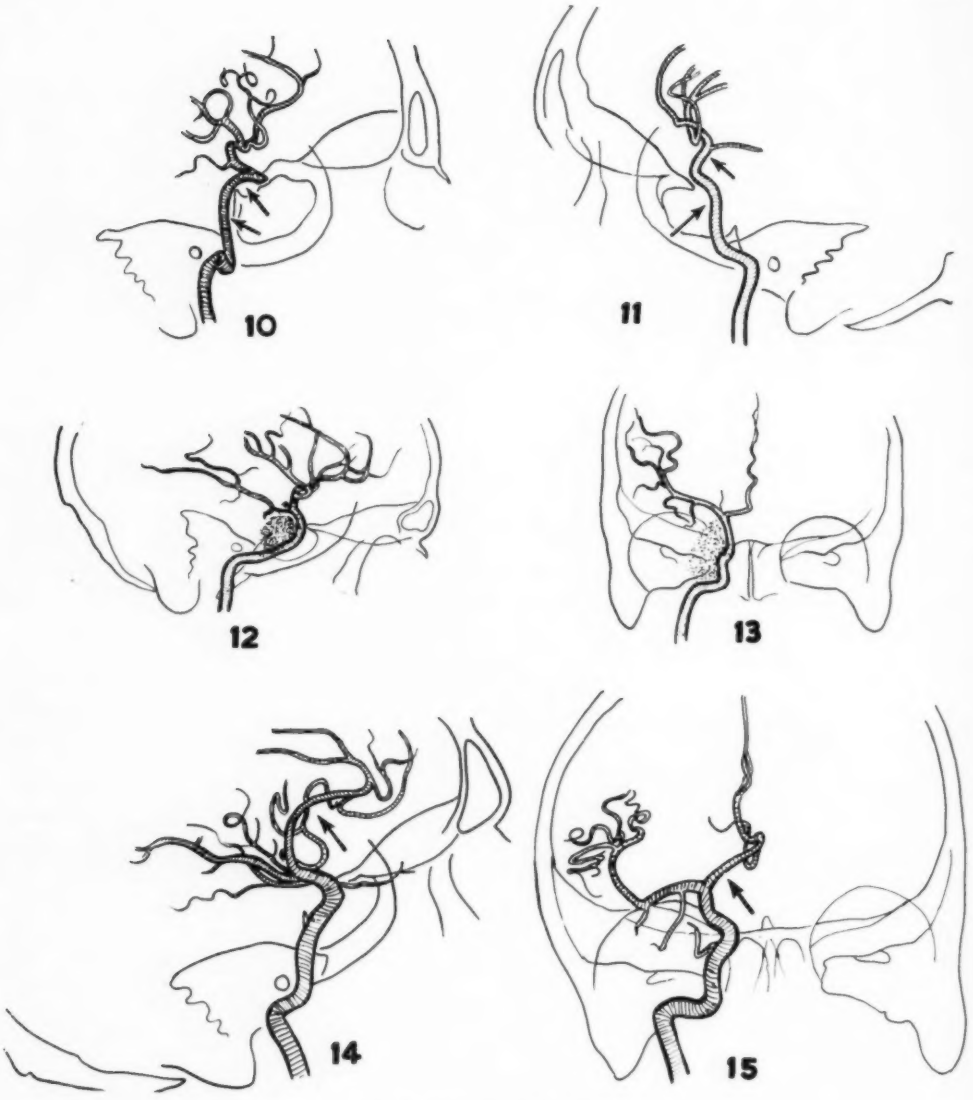
2. TUMORS OF THE BASILAR SURFACE OF THE BRAIN

Even small tumors at the base of the brain can be diagnosed, as a rule, by clinical signs and simple roentgenography of the skull. The main reason for resorting to angiography in these cases is the necessity of differentiation from aneurysms originating from the carotid, basilar, or vertebral arteries or from the circle of Willis. Furthermore, the relationship of the tumor to the large arteries of the base can be well demonstrated by angiography, a significant aid in determining the operability of the lesion. Since many basal tumors attain only moderate size, they displace the adjacent larger arteries without changing the configuration of intracerebral vessels.

(*a*) *Extradural tumors* arising from the *sphenoid bone* (sarcoma), the *sphenoidal sinus* (carcinoma, mucocele), or from the depth of *the sella turcica* (pituitary adenoma) elevate the extradural (subclinoid, parasellar, intracavernous) portion of the internal carotid artery and displace it laterally (Fig. 10); they may compress the vessel considerably and thereby prevent a proper filling of intracerebral vessels (3).

(*b*) *Parasellar tumors*, often meningiomas, open up the first curve of the carotid siphon by flattening and depressing the extradural portion of the carotid artery and by vertical elevation (antero-medial displacement) of the supraclinoid segment of the carotid. Thus, the intradural supraclinoid portion of the carotid appears unusually erect and elongated (Figs. 12 and 13). The angiographic aspect of large parasellar tumors which extend far to the floor of the middle fossa will be described below (see Tumors of the Temporal Lobe).

(*c*) *Suprasellar tumors* (meningioma, adenoma of the pituitary, craniopharyngioma) likewise elevate the supraclinoid portion of the carotid but, in contrast to parasellar tumors, tend to displace the vessel laterally and do not significantly widen the first curve of the carotid siphon. In addition, these lesions characteristically displace the first segment of the anterior cerebral artery upwards and slightly laterally. As a result, the beginning of the anterior cerebral artery forms a downward concave arch above the lesion (Figs 14 and 15). Far advanced pituitary adenomas

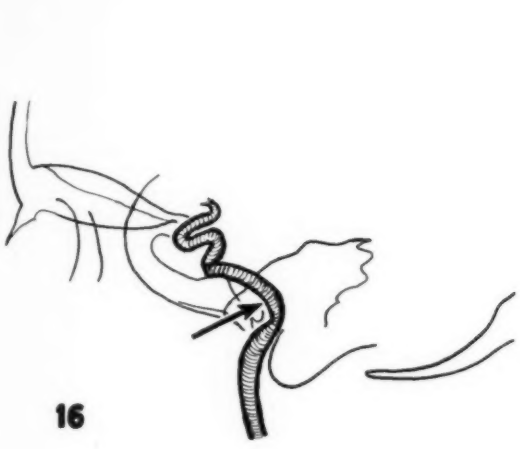


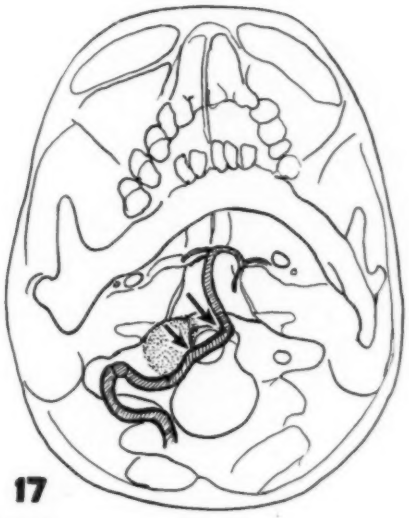
Figs. 10–15. Basilar tumors.

Fig. 10. Lateral arteriogram of mucocele of sphenoid sinus with extradural parasellar extension.
Fig. 11. Lateral arteriogram of pituitary tumor with extradural and intradural parasellar and suprasellar extension.
Figs. 12 and 13. Arteriograms of parasellar tumor with special vascular pattern, lateral and anteroposterior projections.
Figs. 14 and 15. Arteriograms of suprasellar tumor, lateral and anteroposterior projections.

with extradural, parasellar, and suprasellar extension may show a combination of angiographic signs of the three groups described (Fig. 11).

(*d*) *Tumors of the petrous apex and of the retrosellar region* may grow both into the middle and posterior fossa. If the lesion is *extradural, e.g.*, a chordoma, the carotid is compressed and elevated in its extradural retrosellar course (Fig. 16). If the tumor is





Figs. 16 and 17. Basilar tumors.

Fig. 16. Lateral arteriogram of chordoma of petrous apex with extradural retrosellar extension.
Fig. 17. Vertebral arteriogram in basilar projection. Psammoma at anterior rim of foramen magnum.

intradural (meningioma, epidermoid), the angiographic signs are those of a parasellar or basal temporal lobe lesion (see below).

(*e*) It has been stated above that in *tumors of the posterior fossa* ventriculography is the method of choice. In *extramedullary lesions of the 9th, 10th, 11th,* and *12th nerves,* (syndromes of Avellis, Schmidt, Jackson, and Tapia), however, vertebral angiography has some value. First, this method may reveal an aneurysm of the vertebral artery; second, neoplasms in this position are difficult to demonstrate by ventriculography. Vertebral arteriograms should be made both in lateral and basilar projections, since the latter view best brings out the displacement of the vertebral and basilar arteries (Fig. 17).

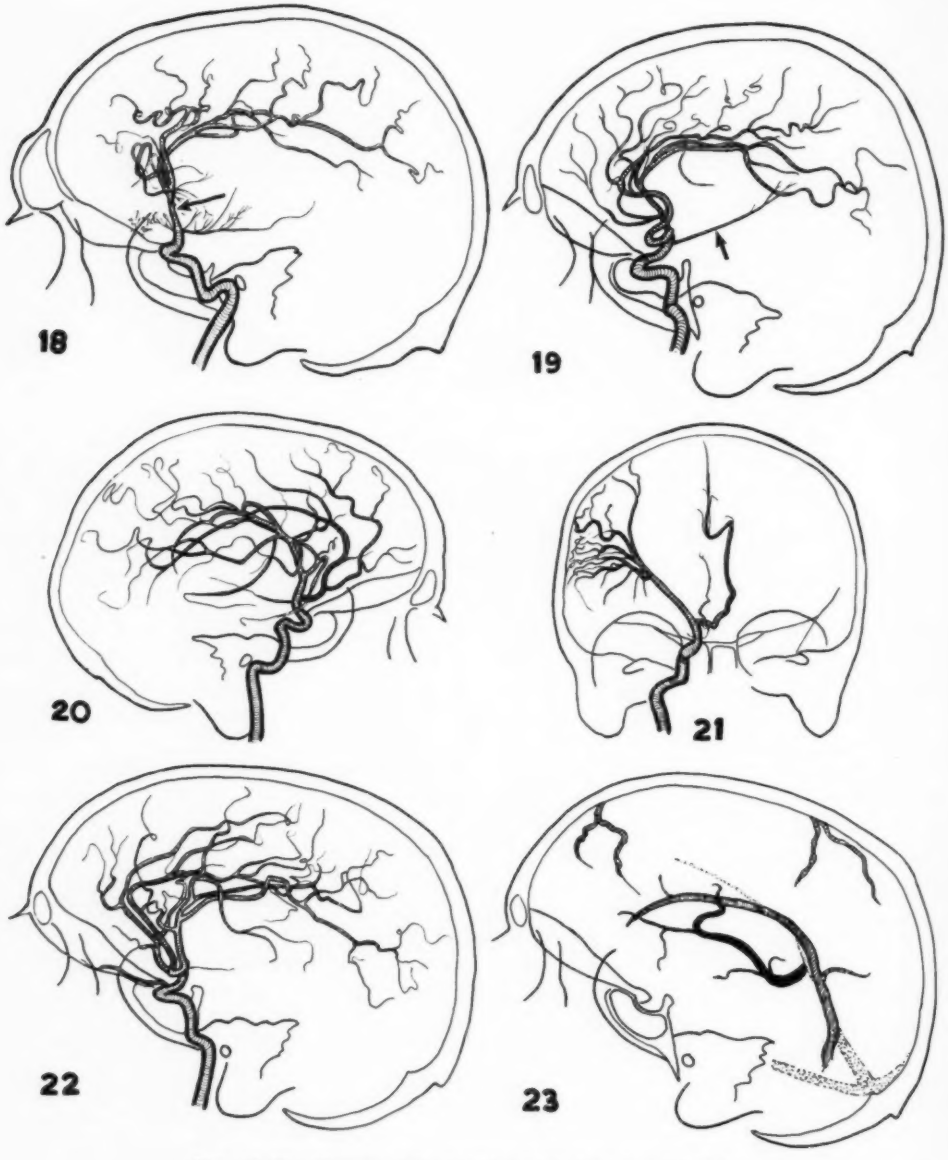
3. TUMORS OF THE TEMPORAL LOBE (2)

The angiographic picture of temporal lobe tumor is so striking that the diagnosis can be made even by one inexperienced in the interpretation of angiograms. Moreover, excellent films, rich in contrast and detail, are easily obtained in these cases.

Elevation of the sylvian vessels is the pathognomonic sign for temporal lobe tumor. In the lateral view, the arteries of the middle cerebral group are displaced upwards; in the anteroposterior projection, they appear also shifted medially, away from the bony contour of the temporosphenoidal convexity. Thus, the distance between the anterior and middle cerebral arteries is reduced in both the sagittal and frontal plane. The anterior cerebral artery is displaced across the midline without losing its normal tortuousness and without deformation of its frontopolar branch. For descriptive purposes, the temporal lobe tumors will be considered under two headings: tumors of the anterior part and tumors of the posterior part.

Anterior Temporal Lobe Tumors: This group comprises meningiomas of the middle fossa arising from the lesser wing of the sphenoid and gliomas of the temporal pole extending to the hippocampal uncus.

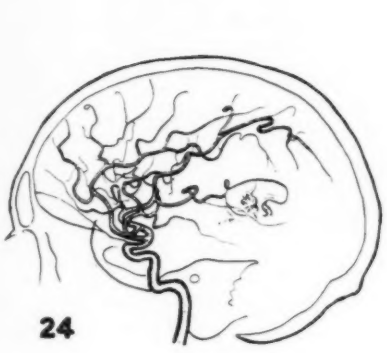
Meningiomas at the inner part of the sphenoidal wing and anterior clinoid process behave like large parasellar tumors and show the corresponding angiographic signs (see above). In addition, they elevate the beginning of the middle cerebral artery. Meningiomas of the external two-thirds of the sphenoidal wing encroach upon the anterobasal part of the temporal lobe and

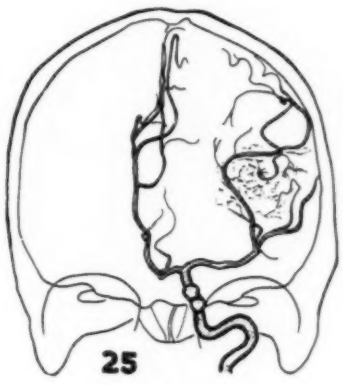


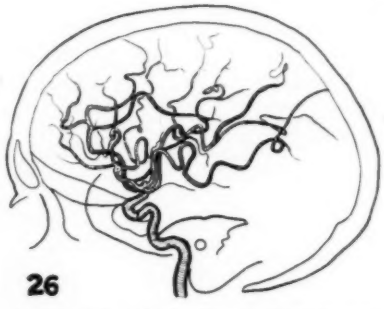
Figs. 18–23. Tumors of the anterior part of the temporal lobe.

Fig. 18. Lateral arteriogram of large meningioma of lesser sphenoidal wing. Arrow points to site of carotid compression.
Fig. 19. Lateral arteriogram of temporal glioblastoma. Arrow points to displaced anterior choroidal artery.
Figs. 20 and 21. Arteriograms of cystic glioblastoma of temporal lobe, lateral and anteroposterior projections.
Figs. 22 and 23. Sarcoma of temporal pole, lateral arteriogram (Fig. 22) and lateral venogram (Fig. 23).

also, to a variable degree, on the postero-inferior part of the frontal lobe. On lateral arteriograms (Fig. 18), these cases show the first curve of the carotid siphon widely open or even flattened to 180 degrees, with the supraclinoid segment of the carotid and the middle cerebral artery taking a straight vertical course upwards. Occasionally







Figs. 24 and 25. Arteriograms of posterior temporal glioblastoma with special vascular pattern, lateral and anteroposterior projections.
Fig. 26. Lateral arteriogram of occipital glioma.

there may be compression or even partial occlusion of these vessels by adjacent neoplasm (Fig. 18). The middle cerebral group then makes a rectangular turn, continuing horizontally in a plateau-like fashion; the terminal branches, however, resume their normal diagonal course. On the anteroposterior projection (Fig. 7), the elongated supraclinoid portion of the internal carotid appears displaced medially; the bifurcation into the anterior and middle cerebral arteries forms a characteristic "V." On venograms, the superficial sylvian vein and the anterior segment of the anastomotic vein of Labbé may be sharply elevated. Gliomas of the anterior part of the temporal lobe exhibit a similar picture (Figs. 20 and 22), although the stretching of the carotid and middle cerebral artery is not as extreme and the backward turn of the sylvian group is more gradual and rounded. Small superficial vessels, the anterior temporal artery or forward branches of the posterior temporal artery, may be stretched over the convexity of the lobe (Fig. 20). If the tumor invades the hippocampal region, the anterior choroidal artery, a fairly constant direct branch of the internal carotid, is considerably stretched and displaced medially and upwards (Fig. 19). In anteroposterior arteriograms, again the typical V-shaped angle between the anterior and middle cerebral arteries may be observed (Fig. 21). On venograms, the engorged vein of Labbé is elevated in a wide semicircle and may project above the level of the vein of Galen, instead of below, as normally (Fig. 23).

Posterior Temporal Lobe Tumors: Posterior temporal lobe tumors, most of which are gliomas, do not deform the carotid siphon and the first segment of the middle cerebral artery, but they markedly elevate the branches of the sylvian group. On lateral arteriograms (Fig. 24) the entire sylvian group forms an arc whose upward

convexity may lie almost at the level of the pericallosal artery. Tumors situated close to the lateral surface of the lobe produce stretching and separation of the angular and posterior temporal arteries and their branches. In the anteroposterior projection, the position of the carotid remains unchanged and the bifurcation into the anterior and middle cerebral arteries has the configuration of a rectangular compressed "U" (Fig. 25).

4. TUMORS OF THE OCCIPITAL LOBE

Tumors of the occipital lobe and adjoining posterior inferior part of the temporal lobe show less characteristic angiographic signs, because these regions receive most of their supply of blood from the vertebral system through the posterior cerebral artery, and that vessel is only occasionally demonstrated by carotid injection.

In lateral arteriograms, moderate elevation and forward crowding of the angular and posterior temporal arteries may be seen (Fig. 26). The terminal branches of these vessels are definitely spread and distended, as are the arborizations of the posterior cerebral artery, provided that vessel is filled. More anteriorly, the compressed arteries of the sylvian group exhibit a tendency to coil together. On the anteroposterior projection, the midline shift of the anterior cerebral artery is slight or even absent. The vein of Labbé may be shifted downward and anteriorly.

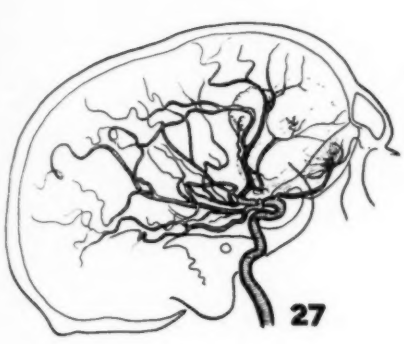
5. TUMORS OF THE FRONTAL LOBE (2, 5)

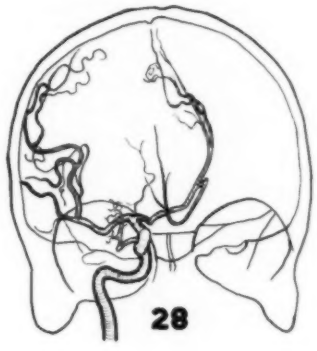
Space-occupying lesions of the frontal lobe deform to a variable degree the supraclinoid segment of the internal carotid artery, the anterior two-thirds of the anterior cerebral artery and its branches, and the beginning of the middle cerebral artery with its frontal ascending (rolandic) branches. The most characteristic sign of all unilateral frontal tumors is the considerable displacement and localized stretching of the anterior cerebral artery to the opposite side. As a rule, this midline shift is more marked in frontal neoplasms than in hemispheral tumors of any other location.

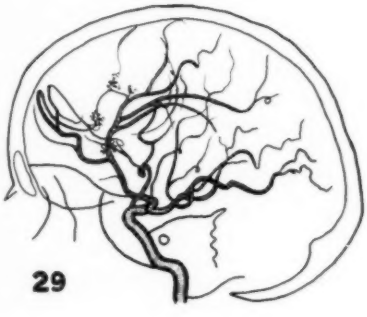
For the purpose of angiographic description, the expanding lesions of the frontal lobe will be divided into the following groups: (*a*) subfrontal (basofrontal) tumors; (*b*) prefrontal (polar frontal) tumors; (*c*) premotor (posterior superior frontal) tumors; (*d*) frontotemporal (posterior inferior frontal) tumors.

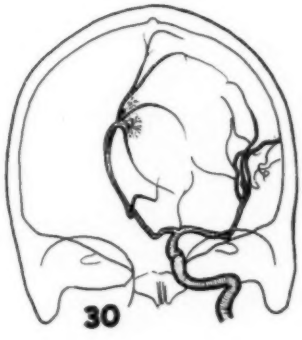
(*a*) *Subfrontal Tumors:* Representative of the subfrontal group are the meningiomas of the olfactory groove. These usually large neoplasms arise from the floor of the anterior fossa and compress the orbital surface of the frontal lobe and suprasellar region; they show more often bilateral than unilateral development. On arteriograms taken in lateral projection, the suprasellar and subcallosal segments of the anterior cerebral artery are displaced upwards and posteriorly, forming an anteriorly concave arc around the lesion (Fig. 27). In large tumors, the supraclinoid portion of the carotid and the precallosal knee of the anterior cerebral artery may be pushed backwards. Unilateral olfactory groove meningiomas displace mainly the subcallosal portion of the anterior cerebral artery to the opposite side (Fig. 28).

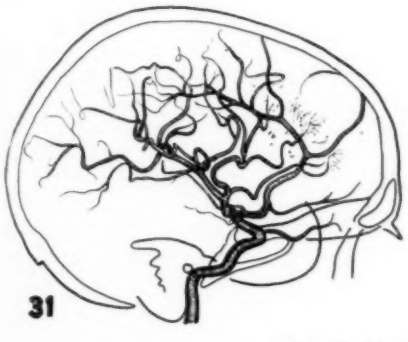
(*b*) *Prefrontal Tumors:* This group comprises mainly astrocytomas and meningiomas (arising from the anterior portion of the falx and superior longitudinal sinus). On lateral arteriograms (Figs. 29 and 31), the carotid siphon and the beginning of the sylvian vessels are slightly pushed backwards and downwards. The anterior cerebral artery is displaced backwards so that the curve of the vessel around the knee of the corpus callosum is blunted without losing its forward convexity. The smaller forward branches of the anterior cerebral artery appear distended and spread apart. Much more impressive changes are observed in anteroposterior projection (Fig. 30). There is major displacement of the anterior cerebral artery across the midline underneath the free edge of the falx and, in contrast to most other hemispheral tumors, the distance between the anterior and middle cerebral arteries is greatly increased. The subcallosal and precallosal

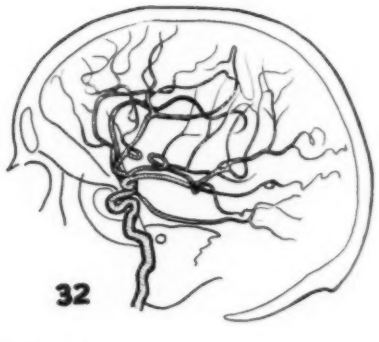












Figs. 27–32. Frontal lobe tumors.

Figs. 27 and 28. Arteriograms of sarcomatous olfactory groove meningioma, lateral and anteroposterior projection.

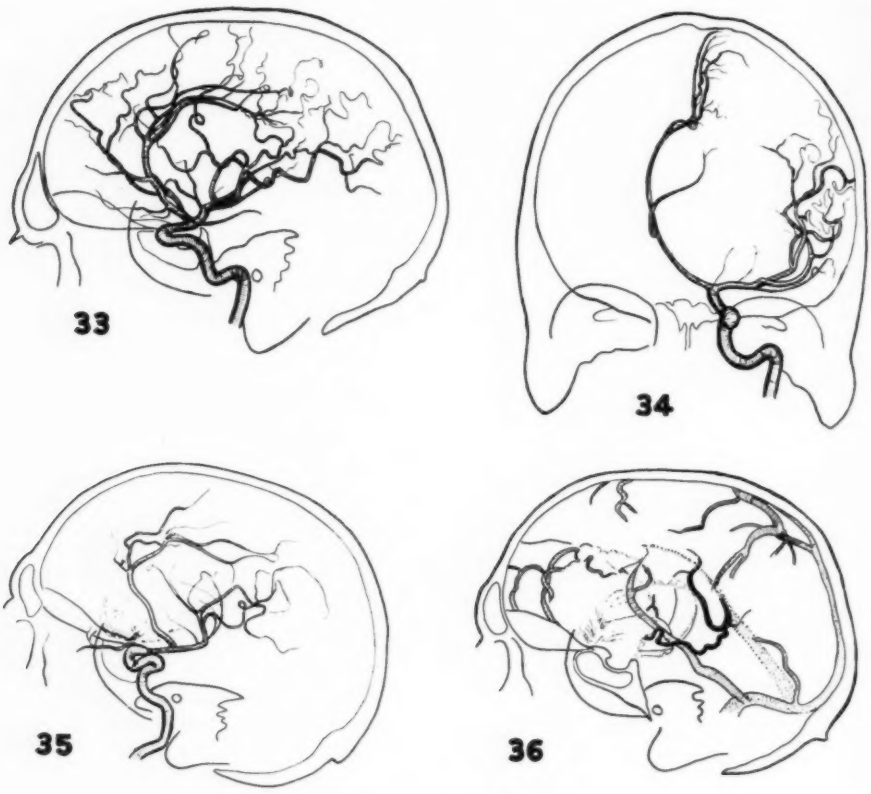
Figs. 29 and 30. Arteriograms of parasagittal meningioma of the prefrontal and premotor regions, lateral and anteroposterior projections. Note special circulation of tumor.

Fig. 31. Lateral arteriogram of astrocytoma of prefrontal lobe. Note faint special circulation.

Fig. 32. Lateral arteriogram of parasagittal premotor glioma.

portions of the anterior cerebral artery form a smooth round bulge and, due to stretching, the extended frontopolar branch leaves the main vessel without undulations. Diagrammatically, the figure outlined by the carotid bifurcation, anterior and middle cerebral arteries resembles an open "O".

(*c*) *Premotor Tumors:* Astrocytomas, oligodendrogliomas, and meningiomas are commonly encountered in this location.



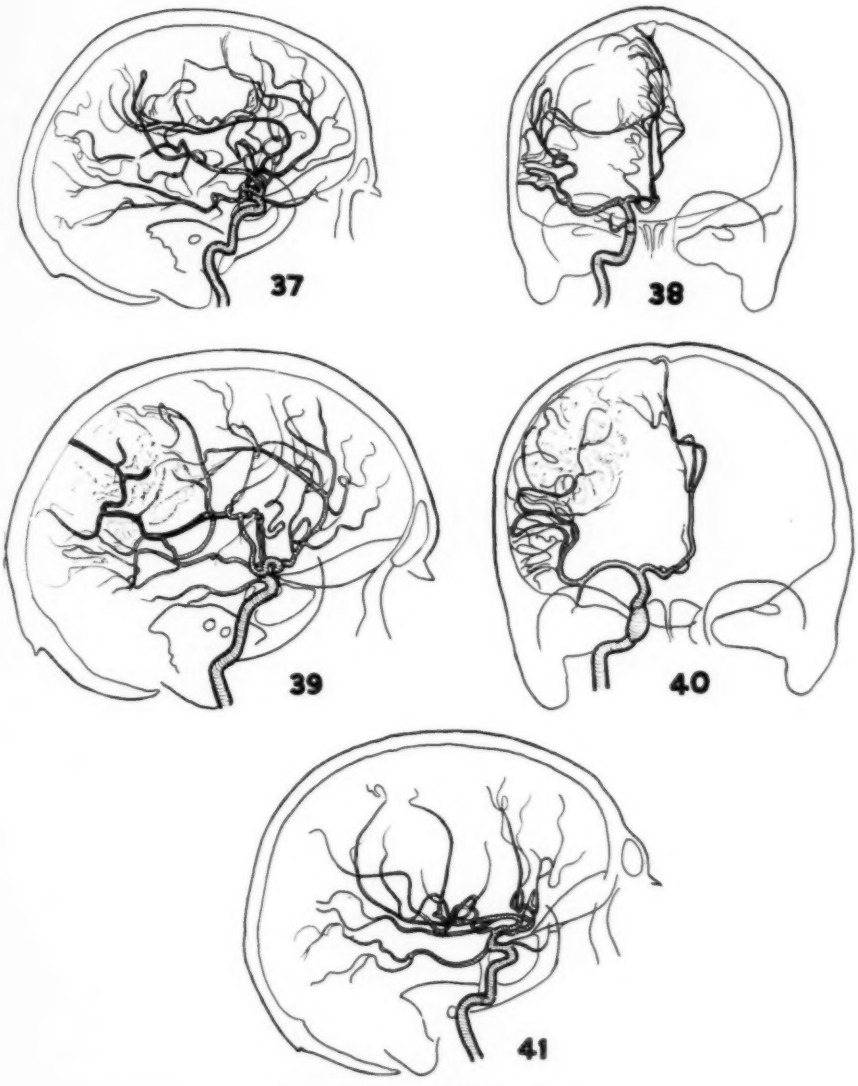
Figs. 33–36. Frontal lobe tumors.

Figs. 33 and 34. Arteriograms of deep posterior and inferior frontal glioblastoma, lateral and anteroposterior projections.

Figs. 35 and 36. Meningioma of lesser wing of sphenoid extending under frontal lobe and beginning of sylvian fissure, lateral arteriogram (Fig. 35) and lateral venogram (Fig. 36). Note special vascular pattern.

The carotid siphon, as seen in the lateral projection (Fig. 6) is compressed, sometimes forming an apparent sharp angle anteriorly. Instead of following a normal diagonally ascending direction, the initial portion of the sylvian group is depressed, taking a horizontal or even downward convex course. It is important to observe the configuration of the ascending frontoparietal artery. This vessel, which has roughly the shape of the Greek letter *psi* (ψ), is difficult to trace in the normal brain, because of its redundancy. In premotor tumors, the frontoparietal artery is displaced downward and posteriorly, with its terminal branches separated (Fig. 32). The anterior cerebral artery may be depressed in its supracallosal segment and the callosomarginal branches separated if the lesion is in parasagittal location, but the polar vessels show little or no spreading. The angiographic picture revealed by the anteroposterior projection is similar to that of prefrontal tumors, with the exception that the principal midline shift occurs a little higher, at the knee of the anterior cerebral artery, which is projected just below the edge of the falx.

(*d*) *Frontotemporal Tumors:* In this subdivision one observes meningiomas of the lesser sphenoidal wing growing underneath the frontal lobe into the beginning of the sylvian fissure, glioblastomas of the posterior inferior frontal and opercular regions,



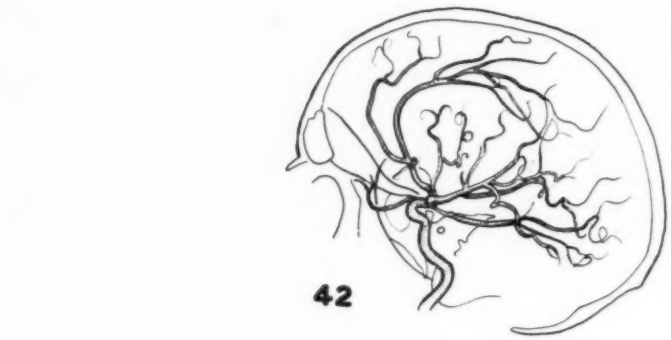
Figs. 37–41. Parietal lobe tumors.

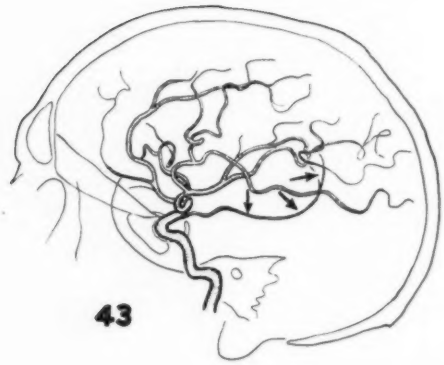
Figs. 37 and 38. Arteriograms of parasagittal meningioma of the anterior parietal region, lateral and anteroposterior projections.

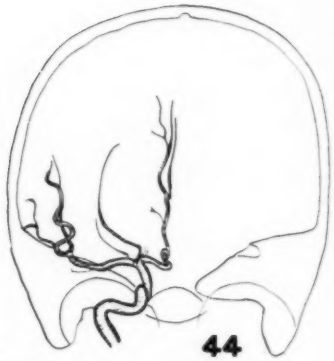
Figs. 39 and 40. Arteriograms of posterior superior parietal glioblastoma with special vascular pattern, lateral and anteroposterior projections.

Fig. 41. Lateral arteriogram of inferior parietal glioblastoma.

and diffuse astrocytomas. In the lateral projection (Figs. 33 and 35), the supraclinoid portion of the carotid forms with the middle cerebral artery a straight, diagonally ascending line. The sylvian group continues in the same direction, or is as often a little elevated as depressed. The ascending frontoparietal artery is markedly stretched, and its two widely separated terminal branches form a triangular pattern. Due to the stretching of the sylvian vessels, the origin of the anterior cerebral artery is projected clear from superimposed vascular shadows. The pericallosal curve







Figs. 42–44. Tumors of the basal ganglia and thalamus.

Fig. 42. Lateral arteriogram of cystic glioma of basal ganglia and thalamus.
Figs. 43 and 44. Arteriograms of glioblastoma of thalamus, lateral and anteroposterior projections. Arrows point to the displaced anterior choroidal artery.

of the anterior cerebral artery is elevated and widened (similar to the picture seen in hydrocephalus). In the anteroposterior projection (Fig. 34), the anterior cerebral artery exhibits the bulge across the midline typical of frontal tumor; the middle cerebral artery may have a tendency to diagonal elevation, as is observed in tumors of the temporal lobe. On venograms, the internal cerebral vein may appear displaced upward and posteriorly (Fig. 36).

6. PARIETAL LOBE TUMORS (2)

Space-occupying lesions of the parietal lobe depress the sylvian vessels and the pericallosal artery. Thus, the distance between the anterior and middle cerebral systems appears to be reduced in the lateral projection (Figs. 37) There is only moderate midline displacement of the posterior part of the anterior cerebral artery because the resistant falx yields little to pressure; yet there may be characteristic notching of the anterior parts of that vessel underneath the falx (Figs. 38 and 40). In parietal tumors, we have relatively often observed poor filling of the terminal cortical arteries. Presumably, the intravascular pressure of the injection is insufficient in the smaller branches to overcome the compression exerted by the tumor.

Neoplasms of the superior parietal region (parasagittal meningiomas, gliomas) (Fig. 37) depress and sometimes separate the pericallosal and callosomarginal arteries. The vessels of the sylvian group may not be much deformed, except for separation of the terminal branches of the posterior parietal and angular arteries (Fig. 39). Inferior parietal tumors, commonly glioblas-

tomas, depress considerably the sylvian vessels but displace little or not at all the anterior cerebral artery in the vertical plane. In the lateral projection, the sylvian group appear compressed and take an unusual horizontal or downward convex course instead of the normal diagonal fan-like arrangement (Fig. 41). The terminal branches (anterior and posterior parietal and angular arteries) leave the sylvian group almost at right angles and ascend vertically, being widely separated from each other. On venograms, some separation of the superior cerebral veins may be noted, and the vein of Labbé may be displaced downwards.

7. TUMORS OF THE BASAL GANGLIA AND THALAMUS

It is sometimes difficult to differentiate clinically tumors of the parietal or parieto-temporal regions from those of the basal ganglia and thalamus. These latter deep lesions are unquestionably best demonstrated by ventriculography, yet a presumptive diagnosis of tumor of the basal ganglia or thalamus can be made by angiography when the following findings are present.

There is evidence of moderate hydrocephalus (see page 322), with the anterior cerebral artery remaining in its normal midline position (Fig. 44). In certain lesions of the basal ganglia, however, this vessel describes an unusual forward and upward bulge similar to that observed in posterior inferior frontal tumors (Fig. 42). If the small arteries of the anterior perforate space (lenticulostriate and lenticulo-optic vessels) are visible in the anteroposterior film, they appear stretched and displaced laterally (Fig. 44). The anterior choroidal artery may be seen displaced downwards and posteriorly, describing a semicircle around the optic tract and pulvinar (Fig. 43). This is almost a pathognomonic sign for thalamic neoplasm, but unfortunately the choroidal vessel is not always visible. According to Egas Moniz (4), the internal cerebral vein and great vein of Galen may be elevated in thalamic tumors; however, we have usually found these veins in normal position.

SUMMARY

The displacement of intracranial blood vessels was studied in 136 cases of space-occupying intracranial lesions by means of carotid (or vertebral) angiography.

The method, which has proved to be reliable and safe, is indicated in certain expanding lesions of the base of the brain and skull, and of the cerebral hemispheres, and is of special value for the differentiation of neoplasms and vascular processes.

The general angiographic changes encountered in expanding intracranial lesions and internal hydrocephalus are discussed.

A detailed description of the vascular deformities is given in lesions of the base of the brain and skull, various parts of the hemispheres, basal ganglia, and thalamus.

626 Medical Arts Bldg.
Grand Rapids 2, Mich.

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Pulmonary Adenomatosis[1]

LESTER W. PAUL, M.D., and GORTON RITCHIE, M.D.

Madison, Wis.

PULMONARY adenomatosis in man is a rare disease of the lungs characterized by the development of multiple nodular adenomatous tumors or by a diffuse hyperplasia of the pulmonary alveolar lining cells. Typically the disease spreads throughout both lungs, interfering greatly with the gaseous exchange in the alveoli and eventually causing death, often with a terminal pneumonia and without the occurrence of metastases elsewhere in the body. The lesion has stimulated considerable speculation and interest, chiefly among pathologists, because of its close resemblance histologically to an infectious disease of the lungs which is widely encountered in sheep and to a lesser degree in some other animals.

The present report is made for the purpose of reviewing the pathologic and clinical aspects of the disease, and more particularly the roentgen features, and to report our experiences in four cases, three of which have not previously been recorded in the literature. A fifth case will be discussed briefly because of interesting findings on histologic study.

Writers on the subject of human pulmonary adenomatosis have been impressed by its resemblance to the condition found in sheep and known variously as jaagsiekte, epizootic adenomatosis, Montana progressive pneumonia of sheep, etc. (3, 9, 12). This disease has been endemic in many areas throughout the world and has been the subject of considerable study. There is sufficient evidence to indicate that it is of infectious origin, though the causative organism has not as yet been found. That a virus is responsible is the opinion of those who have made extensive studies.

In 1939, Bonne (3) described a peculiar tumor-like process of the lungs found in a 30-year-old Chinese, characterized by a diffuse involvement of the alveolar walls of most of both lungs and a replacement of the alveolar cells by darkly staining cells in which mitoses were frequently seen. These cells lined the alveolar sacs and often protruded into their lumina forming papillary buds. There was practically no invasive growth or necrosis, and no metastases were present. Bonne noted the close resemblance to jaagsiekte in sheep and commented on the possible relationship. He did not believe the term carcinoma should be applied to the tumor in his case and suggested that it be called carcinosis. Previous to this report, Dungal (4), who had been studying the condition in sheep, had reviewed the literature in a search for cases of adenomatosis in man and found instances reported by Helly (1907), Löhlein (1908), and Oberndorfer (1930). Of these, the one reported by Helly more closely resembled the condition described. In Oberndorfer's case, metastases were present; Löhlein's patient had a single apple-sized tumor in the lower lobe of the right lung.

In 1943, Sims (9) reported a case which he classified as pulmonary adenomatosis (Case 1 in our series) and also stressed the resemblance to jaagsiekte. He reviewed the entire subject in some detail and concluded that the cases reported by Bonne, Helly, and himself were identical. Subsequently Bell (2), and Wood and Pierson (12) have described quite similar cases and Taft and Nickerson (11) have presented two. For a more detailed discussion and review of the literature, the reader is referred to these articles.

To avoid confusion in the present report, reference will be made only to those cases

[1] From the Departments of Radiology and Pathology, The University of Wisconsin Medical School, Madison, Wis. Read by title at the Thirty-first Annual Meeting of the Radiological Society of North America, Chicago, Ill., Nov. 9–10, 1945.

conforming essentially to the description given above. It should be pointed out, however, that a number of cases of a rather similar nature, histologically, have been recorded, varying chiefly in the fact that metastases have been found, either in the bronchial lymph nodes or disseminated widely throughout the body. Since this particular lesion has shown all of the characteristics of malignancy, it usually has been described as an alveolar-cell carcinoma (6, 10). The clinical course in many of these patients has been somewhat different than in the benign form of the disease, the metastatic involvement often influencing the clinical symptoms and progress to a considerable degree. Neubuerger and Geever (7) made no distinction between the two forms in their review and summarized the records of 24 cases which they believed fell into this category, which they termed alveolar-cell tumor. It has seemed better to us to limit our discussion to those lesions more closely approaching the descriptions given by Bonne and Sims. This does not deny the similarity of origin of a benign and a malignant form but does imply a possibly different clinical progression and a somewhat different end-result on pathologic study of autopsy material. Borderline cases, such as the one described by Sweany (10) are difficult to classify. Sweany's patient had a solitary metastatic nodule in one bronchial lymph node. Other cases of this type are mentioned in Neubuerger and Geever's review (7). The occurrence of areas of carcinomatous change in what is otherwise a benign appearing lesion, without demonstrable metastases (Cases I and III), adds further difficulty to attempts at strict classification.

Of interest in this connection is the statement that metastases have never been found in jaagsiekte except in one specimen reported by Aynaud (1). Just what, if any, relationship exists between jaagsiekte and pulmonary adenomatosis in man remains to be determined. Investigations to determine the infectiousness of the disease and the presence of a virus have been few, and thus far fruitless. Indeed, the very difficulty in recognizing the nature of the process prior to microscopic examination of fixed specimens has largely precluded any attempt at such study. Sims, making use of frozen sections at the time of autopsy, realized the similarity of the lesion to that described as occurring in sheep. Attempts at reproducing the lesions in rabbits, monkeys, and guinea-pigs failed. Wood and Pierson had established the diagnosis in their patient prior to death, on examination of a lobe removed for a lung abscess. The patient died after the disease had become generalized. Studies for identification of a virus were under way at the time of their report.

The pathologic lesion consists typically of scattered nodules of consolidation on the cut surfaces of the lungs. These are usually mistaken for patches of gray pneumonia until microscopic examination reveals that they are caused by the localized occurrence of an abnormal lining in the alveoli. This lining is made up of either low or moderately high cuboidal epithelium, the cells of which tend to be slightly pear-shaped and to form papillary projections into the alveolar spaces. Many investigators regard them as being derived from the alveolar covering cells, but there is some evidence, as seen in our cases, that they come from bronchial epithelium. The alveolar walls in these nodules are thickened and edematous, and the connective tissue is increased.

CASE REPORTS

CASE I (previously reported by Sims): C. H., a white male, 42 years of age, complained of a cough of two years duration. A year prior to admission, rheumatism involving many joints developed. For the past year the cough had been productive of a frothy white sputum amounting to about a cupful a day at the time of admission. Other complaints were afternoon fever, progressive weakness, pains across both sides of the chest upon coughing, shortness of breath even on slight activity, anorexia, and considerable gaseous distention. There had been a 40-pound weight loss during the past year.

Physical examination showed the patient to be very weak, coughing frequently, and raising a watery sputum. The skin was hot and dry. The right side of the chest was smaller than the left and expanded poorly. Dullness was present over the right side of

Fig. 1. Case I. Dense consolidation on the right suggesting extensive pneumonia. Scattered areas of density in the left mid and basal lung field. Autopsy showed adenomatosis and pneumonia.

the chest except at the extreme apex posteriorly. Breath sounds were tubular over the right middle lobe and were decreased generally posteriorly. Many fine moist râles were heard at the apex in the right lung and throughout both bases. An extreme degree of clubbing of the fingers and toes was noted.

Urine and blood examinations were essentially negative, as was the blood Wassermann reaction. Two sputum examinations were negative for acid-fast bacilli. The temperature at the time of admission was 100°, but there was progressive rise during the following days to a high of 106°. The patient became increasingly ill and died five days after admission.

Roentgen examination of the chest the day after admission (Fig. 1) showed a soft patchy mottling throughout the left middle and lower lung field, although no large areas of consolidation were present here. The left dome of the diaphragm was smooth and the sulcus was clear. On the right side there was a great deal of soft opacity, almost completely obscuring the aerated lung except for some air in the apex and peripheral base. The peripheral aspect of the diaphragm was visible and the sulcus was clear. The opinion was expressed that the process was an extensive pneumonia with the major involvement on the right.

The *autopsy* findings were as follows:

Gross: In the right lung there was some fine granular fibrinous exudate on the surface of the lower lobe. Fibrous adhesions were present at the apex and between the lobes. The surface was irregularly nodular to the feel, the nodules being quite large. The cut surface in most areas was pinkish yellow, but in the lower lobe there was extensive fine red pinpoint marking. Only small areas of air-containing lung tissue remained. The *left lung* was air-containing except for the posterior part, which showed various grades of consolidation. There was congestion throughout.

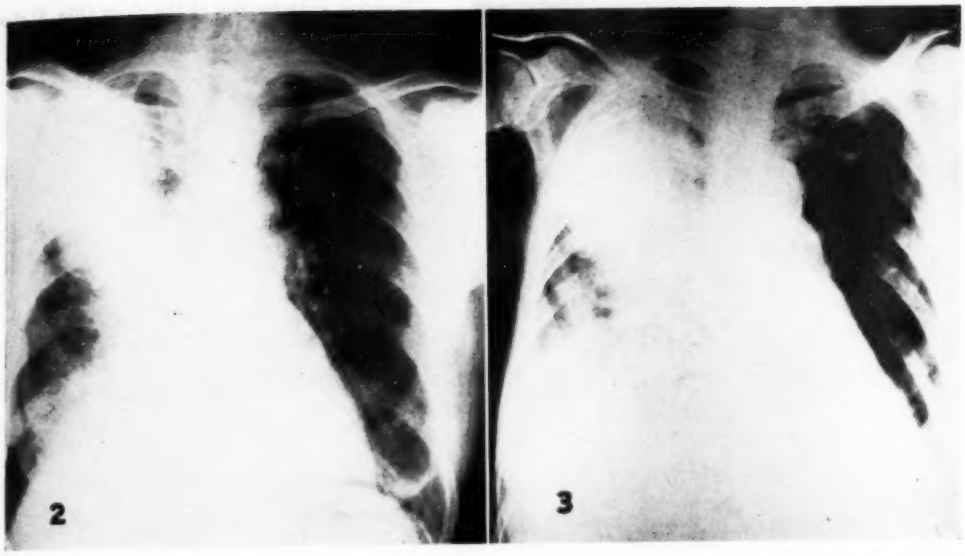
Microscopic: Scattered areas of acute pneumonia were found, and extensive patches of adenomatosis. In some portions this lesion appeared to have undergone malignant change, as there was to be seen an obvious transition between typical adenomatosis and a squamous-cell type of carcinoma.

CASE II: I. B., a 68-year-old white woman, on her first admission to the hospital, on May 11, 1943, had as a chief complaint shortness of breath of six to eight years duration. She had been at bed rest most of the time for the six months previous to admission, with marked shortness of breath on the slightest effort. There was a chronic cough with expectoration of small amounts of frothy white sputum. A weight loss of 40 pounds had occurred in the previous six months. Other complaints included the presence of a lump in the right side of the neck and chronic constipation.

On physical examination enlargement of the thyroid was found. There was a lag in expansion of the right side of the chest, with dullness over the right upper chest anteriorly. Bronchial breathing was present in that area with many fine râles. The heart was enlarged and the rhythm extremely irregular, with a systolic murmur at the apex, transmitted to the axilla. The blood pressure was 162/72. Urinalysis was normal. The blood count showed 14.6 gm. of hemoglobin, 4,350,000 red cells, and 10,400 white cells. The electrocardiogram demonstrated auricular fibrillation and advanced myocardial changes.

Roentgen examination of the chest (Fig. 2) was reported as showing the heart to be moderately and generally enlarged and the aortic arch increased slightly in caliber. There was a good deal of soft truncal accentuation in the lung fields. The right upper lobe was densely consolidated in its inferior two-thirds, and there was homogeneous density running down along the medial lung field toward the base on this side. The sulci were clear. These changes were considered to be due to pulmonary congestion plus pneumonic consolidation on the right. Clinically, there was little to support the diagnosis of pneumonia, and the nature of the right lung consolidation was the subject of considerable speculation. The patient was discharged with the diagnosis of arteriosclerotic heart disease with cardiac enlargement, mitral insufficiency, and auricular fibrillation.

The patient was admitted the second time on Aug. 1, 1943. Her physical condition had deteriorated during the interval. She complained of a great deal of pain over the liver area. Her color frequently became quite cyanotic. Cough had increased and she was raising considerable thin, watery sputum. On



Figs. 2 and 3. Case II. Fig. 2 is the admission roentgenogram. Note the similarity to Fig. 1 (Case I). Three months later (Fig. 3) the disease had undergone a general advance on the right side.

the day of admission, blood in the sputum was noted for the first time. The pertinent physical findings had not changed a great deal as compared with the first admission except for an increase in the chest signs and the presence of a white blood cell count of 32,400 with 93 per cent neutrophils.

Roentgen examination of the chest (Fig. 3) revealed no clearing in the right upper lobe consolidation and a general advance in the process throughout the lung. The evidence of pulmonary congestion persisted. A diagnosis of pulmonary neoplasm was considered the most probable explanation of the right-sided process.

The temperature on admission was 100.2° rectally. The patient failed progressively and died on Aug. 4, 1943, four days after admission.

The *autopsy* findings were as follows:

Gross: In the *right lung*, there were several bronchiectatic abscesses, the largest, in the lower lobe, measuring 7 cm. in diameter. Extensive bronchopneumonia and necrosis were present. The entire lower lobe of the *left lung* was granular to the touch, and scattered over it were nodules about 2 mm. in diameter. Bronchiectasis was also present in the lower lobe.

Microscopic: Extensive acute inflammation and necrosis were found. Scattered over both lungs were areas of adenomatosis of varying sizes up to several millimeters in diameter. The structure of the growth varied greatly in various sections. In some parts it had grown rapidly and extended widely, becoming necrotic locally. In others it was in discrete areas, microscopic in size and sharply limited (Fig. 4). In some of these nodules there were stretches of ciliated columnar epithelium entirely continuous with the remaining cubical epithelium. In the larger nodules there was a definitely invasive tendency, although one would hesitate to render a diagnosis of frank carcinoma.

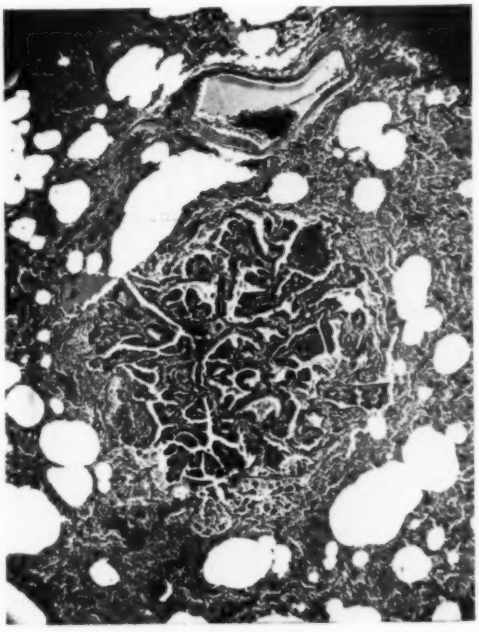


Fig. 4. Case II. A sharply demarcated nodule of adenomatosis. ×*c.* 35.

Fig. 5. Case III. The large mass on the right was a squamous-cell carcinoma. The lesions of adenomatosis were widespread at autopsy but can hardly be recognized as such in the roentgenogram.

An accessory finding of possible interest was a benign polyp of the descending colon, 1 cm. in diameter.

CASE III: G. H., a 76-year-old white male, complained chiefly of inability to walk. Four months prior to admission he stated that he had contracted the "flu," which first affected the respiratory tract and later spread to the gastro-intestinal tract. A month later, pneumonia developed and sulfonamide therapy was instituted. One month before admission there were several attacks of localized pain in the neck and between the shoulder blades. This continued and there developed radiating pain into both arms, occasionally necessitating opiates for relief. Three weeks prior to admission the patient experienced numbness, weakness, and loss of function of the right leg, followed in one week by the same difficulty on the left side. This numbness and paralysis had progressed upward to the abdominal region. There had been, for several years, symptoms suggestive of prostatism. The patient had a loose cough and pain in the chest on coughing.

Physical examination revealed decreased hearing bilaterally, slight cardiac enlargement, a blood pressure of 90/50, and a right indirect inguinal hernia. Neurological examination showed weakness of both arms, areflexia in the abdominal region and both lower extremities, and inability to move either leg. There was loss of sensation below the approximate level of the eighth thoracic segment.

The urine was normal; the blood count showed a hypochromic anemia with a hemoglobin of 11.8 gm., red cells 3,640,000, white cells 13,400.

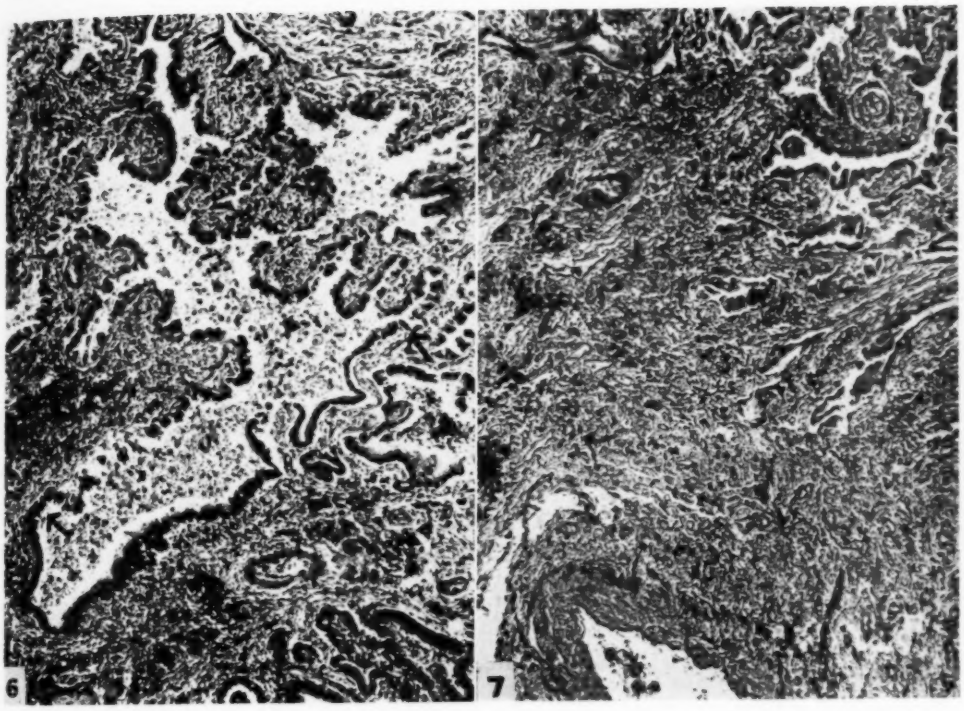
Roentgen examination of the chest (Fig. 5) showed a normal cardiac shadow. The aorta was moderately sclerotic. In the left central lung field was a small amount of fibrosis and a calcified nodule about a centimeter in diameter. There was an apical scar. On the right side was a rounded mass in the central lung field posteriorly, extending from the seventh to the ninth interspace and laterally about two-thirds the distance to the chest wall. In addition, there were irregular fibrous density in the subclavicular region and pleural thickening at the peripheral base, with a partially obliterated sulcus. The impression given was that of scattered areas of tuberculous fibrosis plus a tumor on the right, most likely a bronchiogenic carcinoma. Roentgenograms of the spine showed only some senile osteoporosis and hypertrophic change, and an oil myelogram revealed no lesion. Roentgen therapy was started to the chest and the dorsal spine, but on the sixth day the patient's general condition became so poor that it was discontinued. He became weaker and was unable to expectorate the increased tracheobronchial secretion. Death occurred eight days after admission.

Autopsy revealed the following pertinent findings:

Gross: Right lung: Fibrous adhesions were present, partly obliterating the right pleural cavity. The lower part of the middle lobe and the upper part of the lower lobe were occupied by a large, lobulated, sharply demarcated mass with a necrotic center. The rest of the lung showed localized collapse, edema, and congestion. A large apical scar was present in the upper part of the upper lobe, showing some encapsulated caseous material. *Left lung:* There were localized edema, congestion, and patchy consolidation.

Microscopic examination revealed an extremely varied picture of hyperplasia of the bronchial epithelium. In some areas there was merely overgrowth of this epithelium, with adenomatous structure. In others, there was a rather abrupt transition from ciliated columnar epithelium to somewhat irregular cubical epithelium, and the picture was closely similar to that described as pulmonary adenomatosis (Fig. 6). In apparently continuous transition there was a change in still other areas to frank adenocarcinoma (Fig. 7). Another variation was furnished by the presence in one area (the lobulated mass described above) of a squamous-cell carcinoma, entirely different in structure from the adenocarcinoma. This tumor had metastasized to the local lymph nodes.

CASE IV: F. S., a white male vending machine operator, aged 52, was admitted on July 26, 1945, complaining of shortness of breath of about two years duration. This had become progressively severe during the last seven months. Three months before admission thyroidectomy had been done, without relief. At the time of admission respirations were gasping in character even when the patient lay quietly in bed. Slight effort greatly increased the dyspnea and led to coughing spells, during which a



Figs. 6. and 7. Case III. In Fig. 6, the arrows indicate points of transition between columnar (bronchial) epithelium and the irregular cubical epithelium characteristic of adenomatosis. In Fig. 7, a small area of adenomatosis is seen in the upper right-hand corner, with a transition below to carcinoma. × *c.* 70.

few teaspoonfuls of clear sputum were raised. There had been a weight loss of 45 pounds.

On physical examination the respirations were found to be 50 per minute, the temperature 99.4°, and the pulse rate 128. The patient became cyanotic during fits of coughing. Examination of the chest revealed bronchial breathing throughout both sides with fine râles and dullness in the lower thirds.

The laboratory examinations included a negative serologic test and urinalysis; hemoglobin was 14.8 gm., red cell count 4,600,000, white cell count 11,000. No tubercle bacilli were found in the sputum or gastric contents and the tuberculin test was negative.

Roentgen examination of the chest (Fig. 8) demonstrated a slight widening of the upper and central mediastinal shadow. Aeration was poor in both lung fields. There were a coarse, stringy density and soft, patchy infiltrations throughout both lungs. The extreme apices were somewhat less involved, although not entirely clear. In differential diagnosis consideration was given to sarcoid, Hodgkin's disease, and carcinomatosis. Subsequent examinations showed only a slight advance in the disease during the following six weeks.

Because of the great respiratory difficulty, nasal administration of oxygen was begun immediately on admission and continued most of the time until death. Roentgen therapy was given to the mediastinum, three doses of 200 r each to an anterior and a posterior field. The course in the hospital was almost steadily retrogressive, and death occurred on Sept. 18, 1945, about seven weeks after admission.

The *autopsy* findings with reference to the lungs were as follows:

Gross: There was induration of the right upper lobe, the surface of which had a nodular appearance. Areas of consolidation were scattered throughout the right lung. In the *left lung* generalized congestion was present, with patchy areas of consolidation.

Microscopically there were extensive edema and patchy areas of acute and organizing pneumonia. Many tumor alveoli were seen, lined with cubical epithelium. These appeared in some areas to be entirely benign, while in others there was much more active growth giving the impression of malignancy.

CASE V: G. G., a 60-year-old male, entered the hospital on Oct. 20, 1943, with complaints referable only to the abdomen. He had had a gallbladder drainage one and one-half years before, with relief of symptoms for one year. Lately there had occurred jaundice and a return of right upper quadrant pain.

There were no complaints referable to the chest. A cholecystostomy was done on Oct. 27, 1943, but the patient did poorly and died on Nov. 3.

A roentgenogram of the chest revealed nothing abnormal except some adhesions in the left costophrenic sulcus.

At autopsy, a carcinoma of the pancreas was found, with metastases to the liver, gallbladder, and adjacent areas. In the lungs there were bronchiectasis, atelectasis, emphysema, and acute and organizing pneumonia. One small localized area, found only on microscopic examination, showed obvious benign proliferation of bronchial epithelium. The other autopsy findings were not relevant.

DISCUSSION

The histologic study of these cases brings out two points of special interest, illustrated conspicuously in Cases I, II, and III. In Cases II and III there was an intimate relation between the bronchial epithelium and the cellular lining of the adenomatous alveoli. In Case II there were several stretches of ciliated columnar epithelium among the lower cuboidal cells of the adenomatosis. In Case III an abrupt transition from bronchial epithelium to the adenomatous type of lining was seen in spots, as well as a considerable area of hyperplasia of what was obviously bronchial epithelium. It appears from this that the abnormal epithelium of adenomatosis is derived, in some cases at least, from bronchial epithelium.

The second feature of importance is the obvious transition in Cases I and III from adenomatosis to carcinoma. This would indicate that adenomatosis constitutes in certain cases a transitional form between normal lung and carcinoma, and must, therefore, be regarded as a potentially precancerous lesion.

A study of the records and roentgenograms of our own cases and of those available in the literature was made in order to obtain a more comprehensive picture of the disease. In a few instances, the lesions of adenomatosis were found more or less incidentally at autopsy, the patients dying of some other ailment. In the majority, however, pulmonary adenomatosis was the primary disease. The onset usually was insidious, although in several instances it was accompanied by symptoms of an acute respiratory infection. The two most prominent symptoms were dyspnea and cough, encountered in all cases.

Dyspnea was the first and most distressing symptom in 5 of the 9 cases reviewed and it developed in the remainder during the later stages of the disease. In one of our cases it began six to eight years before admission to the hospital, gradually increasing in severity until the patient finally became bedridden. In another of our patients, dyspnea had been present for two years, becoming more severe in the last seven months; at the time of admission even the effort of talking or of turning over in bed produced severe dyspnea and cyanosis. The reason for the shortness of breath is clear on study of histologic specimens. The widespread alteration of the alveolar walls and replacement of the alveolar cell lining by abnormal cells interferes greatly with the normal exchange of gases. The accumulation of tenacious secretions in the alveoli and bronchi offers an additional hindrance to normal respiration. The dyspnea may be out of proportion to the extent of pulmonary involvement that can be demonstrated roentgenographically. An explanation for this is also found in the study of specimens removed at autopsy. Changes in the alveolar walls may be much more widespread than would be surmised from the roentgenograms. The abnormal cell proliferation lining the alveolar walls is sufficient to prevent adequate gaseous exchange but may not produce much alteration in the roentgenographic pattern of the lungs until complete blockage of the alveoli occurs either due to tumor proliferation or to accumulation of mucus or exudate.

The second symptom common to all was cough. This occurred as an early and prominent complaint in most of the cases reviewed. The cough was productive in all cases, and the sputum invariably is described as thin, watery, frothy, mucoid, and the like. In most instances the sputum was abundant early in the disease, and in the others it became so in the later stages. Frank hemoptysis did not occur, and blood-

streaked sputum was seen only during the terminal stage, when infection may have been a factor in its production (hemorrhagic pneumonia). The expectoration of large amounts of mucoid sputum also has a sound basis as revealed by the study of gross and microscopic specimens and can be explained by the tendency of the abnormal cells to produce mucus. Mucus production is said to be have been less pronounced in those cases which have shown metastases.

four months, and two years, respectively. This corresponds fairly well with cases reported by others.

A review of the roentgenograms of our cases, those reproduced in the literature, and the descriptions given in the remainder reveals two main types of shadow formation, neither of which, by itself, is in any way characteristic. In one group the lesions are manifested by an area or areas of homogeneous density resembling in most features

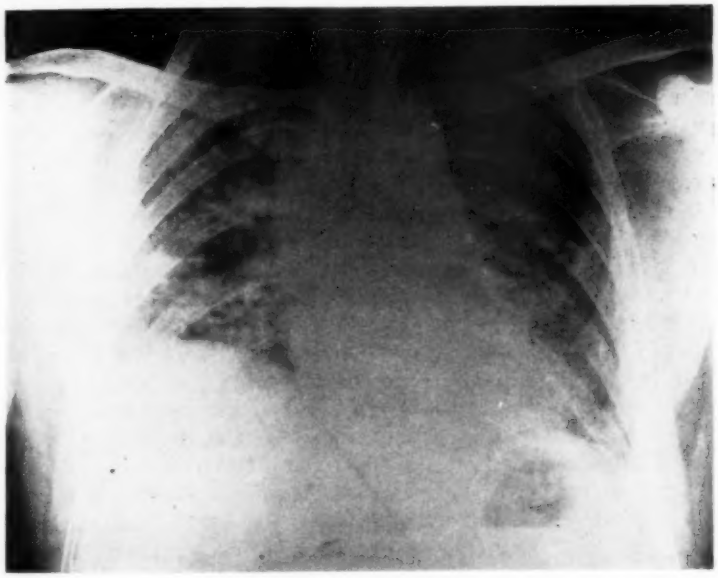


Fig. 8. Case IV. Widespread patchy, infiltrative densities throughout both lungs, with general loss of aeration. Fluoroscopically, limited movement of the diaphragm and poor cardiac pulsations were noted. Autopsy revealed adenomatosis and some pneumonia.

The remaining symptoms—loss of weight and strength, fever, anorexia, etc.—do not appear to have any diagnostic importance since they are common to many diseases.

The number of cases available for study is too small to determine the age and sex incidence with any degree of accuracy. Most cases have occurred in middle-aged and elderly persons, in other words, in the carcinoma-age period. The duration of the disease has generally been a matter of months or even years. In our four cases the duration of symptoms from the time of onset to death was two years, seven years,

that caused by pneumonic consolidation (Figs. 1 and 2). These areas of density do not, as a rule, follow a lobar distribution. They often are bilateral and extensive and may involve most of the lung fields. Signs of atelectasis have not been a prominent feature. If the patient is first seen during the terminal stage of the disease, the diagnosis of pneumonia is more likely to be made, since the clinical signs of infection are encountered frequently at that time. Earlier, the diagnosis of bronchiogenic carcinoma might well be entertained if the involvement is limited to one lung or a part

of the lung. The absence of atelectasis might be of value in differential diagnosis, since it is of such frequent occurrence in bronchiogenic neoplasms.

The second form of the disease is manifested, roentgenologically, by the presence of nodular or patchy infiltrative shadows, again often widely disseminated throughout both lungs. The nodules are not sharply circumscribed but have a hazy outline and resemble in many ways the lesions of widely disseminated carcinomatous metastases. Confusion with metastases is likely, and the two can hardly be differentiated solely on the character of the roentgen shadows in the lungs. The lesions of sarcoid also may resemble this form of pulmonary adenomatosis. Pulmonary sarcoid, however, usually is a benign disease as compared to adenomatosis, the patients, often showing little in the way of constitutional reaction in spite of widespread pulmonary changes. In those cases in which the disease does progress and eventually cause death, the distinction would be difficult to make on roentgen evidence alone. Hodgkin's disease occasionally causes widespread infiltrations in the lungs without much mediastinal lymphadenopathy. If there are no peripheral nodes available for biopsy, a trial of roentgen therapy would seem to be indicated and should prove of considerable value in differential diagnosis. This was done in Case IV of our series. It should be possible to exclude silicosis on the basis of the history and the character of the clinical complaints. The type of lesion under consideration is not ordinarily produced by tuberculosis, but, since the clinical picture may suggest it, studies for tubercle bacilli may be necessary in order to exclude it with reasonable certainty. The same is true for fungus infections.

A combination of massive areas of consolidation and nodular infiltrations has been met with in some cases, changes which are likely to be interpreted as carcinoma of the lung with metastases.

In summary, there is little in the single roentgen observation of a patient with pulmonary adenomatosis that can be considered characteristic of the disease. The shadows produced can be caused by a number of other conditions, and the outlook for eventually establishing definite roentgen diagnostic criteria seems remote. Correlation of the clinical picture with the roentgen evidence may, however, give a clue as to the presence of the disease. Given a middle-aged or elderly patient complaining of severe dyspnea not readily explainable on other grounds, who has a productive cough with abundant thin mucoid sputum and no blood, who shows a slow progressive decline over a period of months, with roentgen changes in the chest such as have been described above, pulmonary adenomatosis should certainly be among the diagnostic considerations. At the present time such a diagnosis is of purely academic interest, since the disease has been fatal in all cases.

Neubuerger and Geever (7) suggest that lobectomy or pneumonectomy might be indicated if the disease could be recognized early enough before it had spread widely. This was done in the case reported by Wood and Pierson, but the patient succumbed to the disease, with the lesions becoming generalized.

The main interest in earlier recognition, or at least suspicion, of this disease would seem to lie in the direction of aiding efforts at determining whether a virus is responsible, establishing the relationship to jaagsiekte in sheep, and in related studies. If the disease is to be recognized prior to autopsy, roentgen examination will play some part in its detection. It is to be hoped that, with the accumulation of more cases in the future, a better understanding of the clinical and roentgen aspects will be available.

NOTE: Since the completion of this paper we have seen an additional case of pulmonary adenomatosis in which the diagnosis was made correctly prior to autopsy on the basis of the clinical and roentgenologic criteria given above. This will be the subject of a later report.

University of Wisconsin Medical School
Madison, Wis.

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Retrograde Abdominal Aortography

A Contribution to the Study of the Abdominal Aorta and Iliac Arteries[1]

PEDRO L. FARIÑAS, M.D.

Habana, Cuba

PLAIN X-RAY examination of the abdominal aorta and iliac arteries offers information only in the presence of calcification of the arterial walls or calcified aneurysms. Retrograde abdominal aortography is a simple method for the study of all the pathological changes of the aorta and iliac arteries. This method consists in exposure of the femoral artery by blunt dissection under local anesthesia at the level of Scarpa's triangle and its puncture with a trocar 1.5 mm. in diameter, through which are injected 50 c.c. of a 70 per cent solution of diodrast in two and a half to three seconds. A tourniquet is applied at the root of each lower extremity in order to prevent the passage of the contrast medium into their arteries. The Trendelenburg position may be required in certain cases.

To avoid changes in pressure when the opaque substance is injected by hand, the author has designed an apparatus that maintains a constant pressure and rate of injection. It consists of a pump with a piston which acts upon the plunger of the syringe. The piston is worked by an air compressor with a regulator and manometer. With a trocar 1.5 mm. in diameter

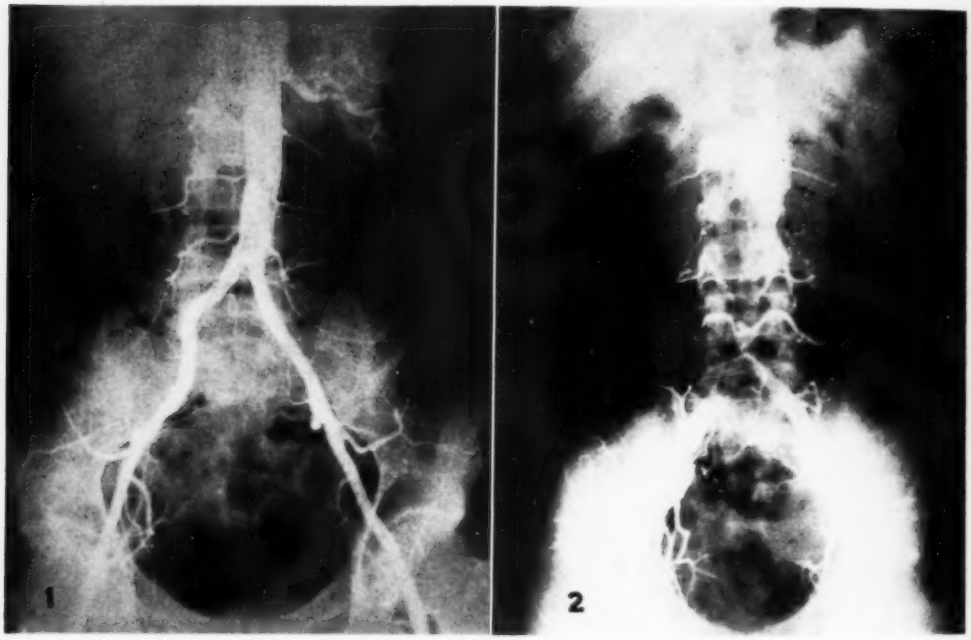

Fig. 1. Uniform dilatation of the abdominal aorta, with a small sacciform aneurysm at the end of the left common iliac.

Fig. 2. Canalized aneurysm of the abdominal aorta and dilatation of the iliac arteries.

[1] Read before the Joint Meeting of the American Roentgen Ray Society and the Radiological Society of North America, Chicago, Ill., Sept. 24-29, 1944.

and a constant pressure of 15 pounds, it is possible to inject 25 c.c. of the opaque substance per second, the speed of injection being modified according to the blood pressure and vascular condition of the patient. The first film is taken when 40 c.c. of the opaque substance have been injected and a second one immediately afterwards, using a fast film changer. When the injection is complete, the trocar is withdrawn, a suture is placed in the adventitia of the artery, and the wound is closed. The patient should recieve a phenobarbital the night before and morphine hypodermically one hour prior to the injection, in order to obtain relaxation and avoid pain.

Fig. 3. Stenosis and dilatation of the abdominal aorta, with narrowing of the iliac arteries and sacculated aneurysm at the end of the common iliac.

Among the inflammatory lesions of the abdominal aorta and iliac arteries, syphilitic arteritis occupies first place because of its frequent occurrence. It may produce dilatation or obliteration of the arteries. In dilatation the muscular and elastic coats of the artery are destroyed, leading to the formation of the different types of aneurysm. As examples we have the following histories:

Fig. 4. Aorta dilated, elongated, and tortuous. Large ovarian cyst.

S. B., white, 52 years old, married, had untreated syphilis; the Kahn and Meinicke tests were markedly positive. The abdominal aorta was palpable, and there was a systolic murmur. Arteriography shows uniform dilatation of the abdominal aorta with a small sacciform aneurysm at the end of the left common iliac (Fig. 1).

R. G., 30 years old, gave a history of slight epigastric pains for six months with no relation to meals; then, sudden epigastric pain of increasing intensity, with radiation toward both lower extremities. The Kahn and Meinicke tests were strongly positive. Palpation revealed a pulsating epigastric tumor the size of a grapefruit. The clinical impression was a tumor of the head of the pancreas. Aortography showed dilatation of the iliac arteries and a large canalized aneurysm of the abdominal aorta. This diagnosis was confirmed surgically, and arteriotomy of both iliac arteries was performed (Fig. 2).

In the obliterative type of arteritis there is a thickening of the arterial wall, especially the intima, with reduction of the

Fig. 5. Reduction of the lumen of the external iliac and complete obstruction of the internal iliac. Greatly increased collateral circulation forming an internal arc (epigastric and internal mammary) and an external arc (circumflex iliac and lumbar arteries).

Fig. 6. Endarteritis obliterans involving all right iliac arteries, reducing their lumen. Marked collateral circulation.

lumen. Thrombosis may cause complete obliteration. The muscular coat undergoes degeneration, leading to dilatation of the vessels. As an example, we have the following case:

R. G., 69 years old, white, with a clinical diagnosis of syphilitic aortic ectasia, gave a history of dyspepsia one year previous to hospitalization. The blood pressure was 205 maximum and 80 minimum; pulse rate 80 per second; oscillometric index, right thigh 2 1/2, left thigh 2 1/2. Abdominal palpation revealed an elongated, firm, pulsating aorta. On auscultation, a systolic murmur was heard. Aortography showed narrowing of the iliac arteries with a sacculated aneurysm at the end of the common iliac, as well as stenosis and dilatation of the abdominal aorta (Fig. 3).

Degenerative lesions are more commonly found at the end of the abdominal aorta and iliac arteries. They originate in the intima and extend to the muscular and elastic coats. The lesions of the intima produce thickening of the walls with narrowing of the lumen; those of the muscular and elastic coats produce atrophy. The vessel then loses its contractility and allows itself to become dilated by the blood pressure. Besides the dilatation, the artery becomes elongated and tortuous. The visceral branches of the aorta may become involved, impairing the circulation of the abdominal organs. The following case shows the aorta dilated, elongated, and tortuous (Fig. 4):

A white woman had a large ovarian cyst. Radiologically, besides the ovarian cyst, we observed a parietal thrombosis of the terminal aorta, as shown by indentations of the arterial wall.

Thrombosis may completely obliterate the lumen of the vessel. As examples we have the following cases:

A. M., 74 years old, white, was hospitalized because of pain, cyanosis, and swelling of the right foot

Fig. 7. Advanced atheroma of the aorta and iliac artieries with almost complete occulsion at the aortic cone.
Fig. 8. Roentgenogram of the anatomic specimen of the same case, showing the calcification and kinking of the arteries.

with ulceration of the fourth toe. There were symptoms of intermittent claudication of approximately a year's duration.

Lately the pain had become intense, affecting both the foot and limb, increasing when the extremity was in the horizontal position. Physical examination revealed cyanosis of the toes. The skin was atrophic, with necrosis of the right big toe, including the interdigital space. This foot had a lower temperature than the other, with a non-palpable arterial pulse. There was marked sclerosis of the peripheral arteries. The arterial blood pressure was 130 maximum, 70 minimum; pulse 75. The oscillometer reading was 0 in all the limbs. Aortography showed marked reduction of the lumen of the external iliac and complete obstruction of the internal iliac arteries. Collateral circulation was greatly increased, forming an internal (epigastric and internal mammary) and an external arc (circumflex iliac and lumbar arteries). Figure 5 shows this collateral circulation, which is believed to be the first such observation in the living. There was thrombosis with complete obstruction of the terminal aorta and iliac arteries. A film made ten minutes after the injection revealed a normal urogram, showing that circulation above the obstruction was unimpaired.

P. P., 54 years old, was hospitalized because of intermittent claudication of ten years duration. Physical examination revealed palpable epigastric pulsation. Palpation of the abdominal aorta and iliac arteries was painful. There was a very faint pulsation of the femoral arteries, especially of the left, with complete absence of pulsation at the left popliteal and posterior tibial arteries. The oscillometer did not record any pulsation in either of the lower extremities. The toes of the left foot became pale when the limb was raised for a few minutes and cyanotic when it was lowered. The blood pressure was 110 maximum, 70 minimum; pulse rate 72. A clinical diagnosis of arteriosclerosis with thrombo-arteritis at the bifurcation of the aorta and iliac arteries was made. Aortography showed an advanced degree of endarteritis obliterans involving all the right iliac arteries and considerably reducing their caliber, with marked collateral circulation (Fig. 6).

In advanced cases, dilatation, elongation, and lime deposits give rise to a characteristic picture. By aortography the author has observed valvular kinks that retard circulation considerably. He believes himself to be the first to demonstrate

them radiologically in the living (Figs. 7 and 8). Surgical removal of these valvular kinks was shown to benefit greatly the circulation of both extremities.

CONCLUSION

The principal inflammatory and degenerative processes encountered by the author in the arteriographic study of the abdominal aorta and iliac arteries are described above. It is hoped that this procedure, which converts aortography into a simple arteriography, will contribute to a better knowledge of the pathological conditions of these vessels, thus offering new approaches to treatment.

Calle 23 No. 411
Habana, Cuba

Bilateral Osteochondritis Dissecans of the Knee[1]

CAPT. R. H. HERMANSON, M.C., A.U.S.
Station Hospital, Fort Leavenworth, Kansas

"OSTEOCHONDRITIS dissecans is an osseocartilaginous lesion of debatable etiology characterized by partial or complete demarcation of a segment of articular cartilage and subchondral bone with or without ultimate detachment and extrusion into the joint (16)."

INCIDENCE

Osteochondritis dissecans occurs most frequently in young adults. King (10) found the average age of onset to be eighteen years. Males are affected four times as frequently as females. Of the reported cases, 85 to 90 per cent involved the knee joint (2, 14, 16). In decreasing order of incidence, the hip, elbow, and ankle are also affected. Hutchison (9) saw one case in the proximal phalanx of the right great toe and one in a metacarpal bone. Among most clinical workers, however, the disease is considered to be primarily one of the knee joint (8).

In a series of 24 cases, King (10) found the condition to be bilateral in the knees in 20 per cent. Bernstein (1) reported three cases involving both knees in members of the same family, one brother and two sisters. King and Richards (11) found the disease to be bilateral in 30 to 35 per cent of a series of cases involving the hip. Richards (15) and Dawson (4) each reported one case of bilateral osteochondritis dissecans of the knee, and others (8, 12, 14) state that both knees may be involved. de Lorimier (5) presented a case in which the lesion involved the patella of one knee and the medial femoral condyle of the opposite one.

Among lesions producing internal derangement of the knee joint, osteochondritis dissecans is third in incidence (3). The commonest site is the lateral aspect of the medial femoral condyle. Occasionally the lateral condyle or the patella may be involved.

ETIOLOGY

The etiology of osteochondritis dissecans remains unknown and no entirely satisfactory explanation has yet been offered (14). The majority of authors favor a traumatic cause. Both intra- and extra-articular trauma have been indicted. Impairment of blood supply to the region affected, either as a result of damage to the blood vessels by the impaction of opposing articular surfaces or avirulent bacterial emboli lodging in epiphyseal arteries, has been considered as an etiologic factor. Dawson (4) considers that the lesion may be due to congenital changes or have a developmental background. Bernstein (1), Harbin and Zollinger (7), and Wagoner and Cohn (17) regard heredity as of importance. Bernstein's cases of bilateral involvement of homologous joints in members of the same family are striking and lend important weight to this view. It may well be that trauma is the inciting cause only in joints that are congenitally predisposed to this lesion. It is significant that no inflammatory changes have been observed in the loose body.

PATHOLOGY

Conway (2) has described three stages in the pathology of osteochondritis dissecans. In the first stage there is only a fairly well demarcated prominence of the articular surface. The articular cartilage covering this elevation differs in color from the rest of the cartilaginous surface and the process can easily be removed at this stage. It may also become permanently re-attached (14).

In the second stage the fragment has become more distinctly separated and lies within an excavated area of the articular surface. It may be held in place by only a

[1] Accepted for publication in November 1945.

thin band of fibrous tissue or by a fairly firm adhesion. While it is attached, the fragment may be nourished by blood vessels in the pedicle and proliferation of a layer of fibrocartilage along the surface of separation may occur.

In the third stage, complete sequestration of the fragment into the joint cavity occurs. The detached fragment may remain free within the joint or become fixed to the synovial wall. When they are free in the joint, there is a pronounced tendency for the loose bodies to migrate into quiet areas, commonly the posterolateral pouch, the subquadriceps bursa, or the anterior compartment of the knee (10). After complete separation, all of the bony portion usually undergoes necrosis (14). The cells of the fibrocartilaginous covering are nourished by the synovial fluid. These cells slowly proliferate and the body increases in size. The growing fibrocartilage gradually absorbs and replaces the necrotic articular cartilage, and more slowly the necrotic bone, so that after a period of years the original constituents of the loose bony fragments may completely disappear. Concomitant calcification in the superficial layers of fibrocartilage produces a pathological type of new bone. Trauma to the articular surfaces produced by impaction of the loose body may result in extensive arthritic change. This usually results after the passage of a long period of time.

For obvious reasons, the changes in the osteochondritic bed have been less well studied. Wolbach and Allison (18) were able to study a case postmortem in a patient dying from another disease. They found a cyst in the cancellous bone beneath the fibrocartilage, to which they attributed etiologic significance in the formation of the loose body. The excavated area gradually becomes filled with fibrocartilage and the outline becomes shallower and less pronounced. In time, it may become exceedingly difficult to demonstrate.

CLINICAL PICTURE

Constant symptomatology is conspicuously absent in osteochondritis dissecans, and a pathognomonic clinical picture is unknown. Acute symptoms occur after the fragment has been extruded from its bed; before this stage there may be no symptoms. According to King (10), patients may be divided into three groups. In the first group there is a sudden onset a few days subsequent to a twist or other injury. No history of previous disability is obtained. The joint is swollen and locked in 15 to 45° flexion. In the second group the condition is asymptomatic and is discovered on roentgen examination of a supposedly normal knee. Patients of the third group complain of a chronically troublesome knee with soreness, pain, and locking. Muscular atrophy is a late finding. It is significant that in King's series the history and clinical examination alone led to the diagnosis in only one case.

ROENTGEN FINDINGS

The diagnosis is readily established by roentgen examination. The osteochondritic focus appears as an arcuate area of diminished density in the medial femoral condyle. Before separation occurs, this may be only a faint shadow outlining a smooth, almond-shaped piece of bone which is of normal density and not appreciably elevated. As the disease progresses, the area of diminished density increases in size, due in great part to the proliferation of fibrocartilage both in the osteochondritic bed and around the separated fragment. Elevation and projection of the fragment beyond the surface of the surrounding normal bone then ensues. Fragmentation of the sequestrum is not unusual, especially with large sequestra. One sequestrum may divide into two or more fragments and present the appearance of cystic degeneration. With necrosis of the bony portion, the density of the fragment increases. The structure and density of the bone about the pathologic process remain normal throughout the course of the disease. In old, longstanding cases, however, arthritis may supervene, with resultant marginal osteophyte formation. After the loose fragment has migrated, recognition of the disease

may be slightly more difficult. Usually a tell-tale depression will remain in the femoral condyle and serve as a beacon for the alert observer.

Special roentgenographic technics are rarely necessary. In the great majority of cases, anteroposterior and lateral views will suffice accurately to delineate the process. Occasionally a postero-anterior projection with the knee in 45° flexion is indicated for better definition of the osteochondritic focus and to visualize the loose fragment within the joint. A very small loose body may sometimes be seen after the injection of air into the joint when other methods have failed to depict it.

DIFFERENTIAL DIAGNOSIS

Confusion with other disease processes seldom exists. Less frequent causes of loose body are synovial osteochondromatosis, trauma (13), and fracture of an osteophyte in osteo-arthritis. In osteochondromatosis the synovial membrane is involved, there are multiple loose bodies, and the underying bone is intact. A history of preceding trauma and the location of the defect on the most exposed parts of the bones will serve to differentiate traumatic loose bodies. When fracture of an osteophyte occurs, other osteophytes usually are present at the articular margins of the tibia and femur, and the loose body usually has a greater density than the loose body of osteochondritis dissecans. Infectious arthritis, either specific or non-specific, may produce sequestration in the underlying bone. The location of the sequestra usually differs from that of osteochondritis dissecans and the regional bone is involved in the inflammatory process. Moreover, in acute infectious arthritis the joint space is increased because of fluid, and after the disease becomes chronic, the joint space is decreased because of the destruction of articular cartilage. The presence of a fabella should rarely cause confusion.

TREATMENT

A detailed discussion of therapy is beyond the scope of this paper. So far as the knee is concerned, there is great unanimity of opinion that surgery is the treatment of choice. Cases without urgent symptoms and without elevation of the fragment may respond well to immobilization alone. Liebman and Iseman (13) report such a case.

CASE REPORTS

Three cases of bilateral osteochondritis dissecans of the knee were recently observed at this station hospital within a period of three months and they are considered sufficiently interesting to merit presentation. During this interval, roentgen examination was performed on 92 knees. In this small series these were the only cases of osteochondritis dissecans observed. While the number is too small to permit of any conclusions, it does emphasize the fact that bilateral involvement is not uncommon. When the disease is found in one knee, routine roentgen examination of the supposedly normal knee should be performed. This procedure will uncover many unsuspected cases and may demonstrate that bilateral involvement is more common than it is at present thought to be.

CASE I: A 20-year-old soldier on active military duty for fourteen days reported to sick call, complaining of pain and stiffness in both knees of twelve years duration, occurring after walking or other exercise. There was no history of injury.

On physical examination both knees were found to be slightly enlarged with some limitation of flexion. There was slight tenderness along the anterior aspects of both medial femoral condyles. No instability was observed.

X-ray examination revealed a loose body about 2.5 × 3.5 × 1 cm. along the lateral aspect of each medial femoral condyle with a correspondingly large excavation of the condyle. The loose body in the right knee was fragmented.

CASE II: A 23-year-old soldier on active military duty for twenty-three months reported to sick call, complaining of inability to perform his duties. He stated that since the age of 13 he had suffered periodically from pain and weakness in the knees; that in civilian life his left knee not infrequently snapped and locked, and that it might take several days to regain full motion. During basic training he had trouble with the knee which necessitated re-assignment to less strenuous duties. After a time, he was returned to training and began to experience diffi-

Fig. 1. Case I. Note the large size of the sequestra and fragmentation in the right knee, which is shown in the upper views. The lower views are of the left knee.

culty with the right knee. This joint never locked, but an occasional clicking sensation was experienced. There was no history of trauma.

Physical examination revealed slight puffiness about both knees but no actual increase in joint fluid. There was a free range of motion, with slight lateral instability of both knees.

X-ray examination revealed an irregular defect in the lateral aspect of each medial femoral condyle with a dense button of bone adjacent to the defect.

Fig. 2. Case III. The upper views are of the right knee and the lower ones of the left knee. Note fragmentation of sequestrum in right knee.

The process was more extensive in the left knee and the sequestrum was fragmented.

CASE III: A 19-year-old private on active duty for two months reported to sick call, complaining of pain in the knees for six years. The pain was sharp and was aggravated by walking and by bad weather. At night there was a mild ache and occasionally slight swelling of the knees. Frequently a gritty sensation was felt in the knees. There was no history of

trauma. The patient's commanding officer noted that he appeared very willing but was unable to drill.

The family history was interesting in that the patient's father had had "rheumatism" of the knees and hands for several years. No x-ray studies had been performed, however, and it could not be determined if he also was suffering from osteochondritis dissecans.

Physical examination revealed no evident abnormality. Both knees were stable and had a full range of motion.

X-ray examination showed a smooth button of bone at the lateral aspect of each medial femoral condyle, with a corresponding arcuate defect in the condyle. The loose body on the right extended for a considerable distance into the joint.

All three patients were considered unfit for further military duty and were discharged from the Army.

SUMMARY

1. Three cases of bilateral osteochondritis dissecans of the knee occurring in young adult males have been presented, and the incidence, etiology, pathology, and diagnosis have been discussed.

2. Disability was sufficient in each case to require separation from the military service.

3. Bilateral osteochondritis dissecans is not uncommon.

4. The desirability of routine roentgen examination of the supposedly normal knee is emphasized, when osteochrondritis dissecans is found in one knee. This procedure may demonstrate that bilateral involvement is more common than is now suspected.

14 Charlesgate West
Boston, Mass.

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Heart Size from Routine Chest Films[1]

PAUL C. HODGES, M.D.

The Division of Roentgenology, The University of Chicago

ROENTGEN RAY determination of the size of the human heart may be extremely precise if one employs sufficiently refined technics, such as those for volume, or a mere approximation if the subjects have heart disease and the criterion of size is the transverse diameter of the frontal plane silhouette. Estimation of frontal plane area from orthodiagrams or from specially made films constitutes a practical compromise between these two extremes, and tables and equations have been developed for predicted normal area from height and weight.

The general radiologist faces daily the task of passing judgment on the shape and size of the hearts that he sees in routine chest films. In our laboratory such films invariably are stereoscopic and are accompanied by a record of the patient's height, weight, and anteroposterior chest diameter. Exposure times range from 1/30th of a second to 1/10th of a second, depending on the thickness of the chest, and there is no attempt to synchronize exposures with a particular point in the cardiac cycle. In spite of this, we find that there is seldom much difference in the size of the heart shadows in the two films. Occasionally one shadow will be distinctly smaller than the other, but in such cases reraying usually yields two shadows approximately alike and approximately the same as the larger shadow in the first pair.

These observations have led us to believe that the systolic size rarely is recorded on routine chest films and that the larger shadow in a pair of such films may be accepted as of approximately diastolic size.

When general inspection suggests cardiac enlargement or there is clinical reason to suspect it, we measure gross frontal plane area from the larger of the two shadows in our chest films and then correct this value for divergent distortion. If the net frontal plane area thus obtained is within plus or minus 10 per cent of the normal area predicted by the patient's height and weight, we report it as normal but add in brackets the actual percentage variation from normal. When the variation exceeds 10 per cent, we report the heart as N per cent over- or undersize.

TECHNICAL DETAILS

With a wax pencil the right and left borders are sketched in as solid lines and the arbitrarily drawn upper and lower limits as broken lines. These lines are now transferred to white tracing paper and, by means of a planimeter, the area enclosed within the solid and broken lines is measured—in other words, the gross frontal plane area of the heart. In our laboratory the outlining and tracing are done by the radiologist, the measurements by stenographers.

CORRECTION FOR DIVERGENT DISTORTION

Because of the divergent distortion that is always present in ordinary roentgenograms, gross area is greater than true or net frontal plane area, the latter being obtained from the former by multiplying gross area by a correction factor F, obtained from the equation:

$$F = \left[1 - \frac{K}{D} - \frac{C}{3D}\right]^2 \qquad (1)$$

where F = divergent distortion correction factor; K = distance in cm. between anterior surface of chest and film (in our apparatus, 2.5 cm.); D = distance in cm. between target of x-ray tube and surface of film (in our apparatus, 183 cm.); C = anteroposterior diameter of chest in cm. (varies

[1] Read by title at the Thirty-first Annual Meeting of the Radiological Society of North America, Chicago, Ill., Nov. 9–10, 1945.

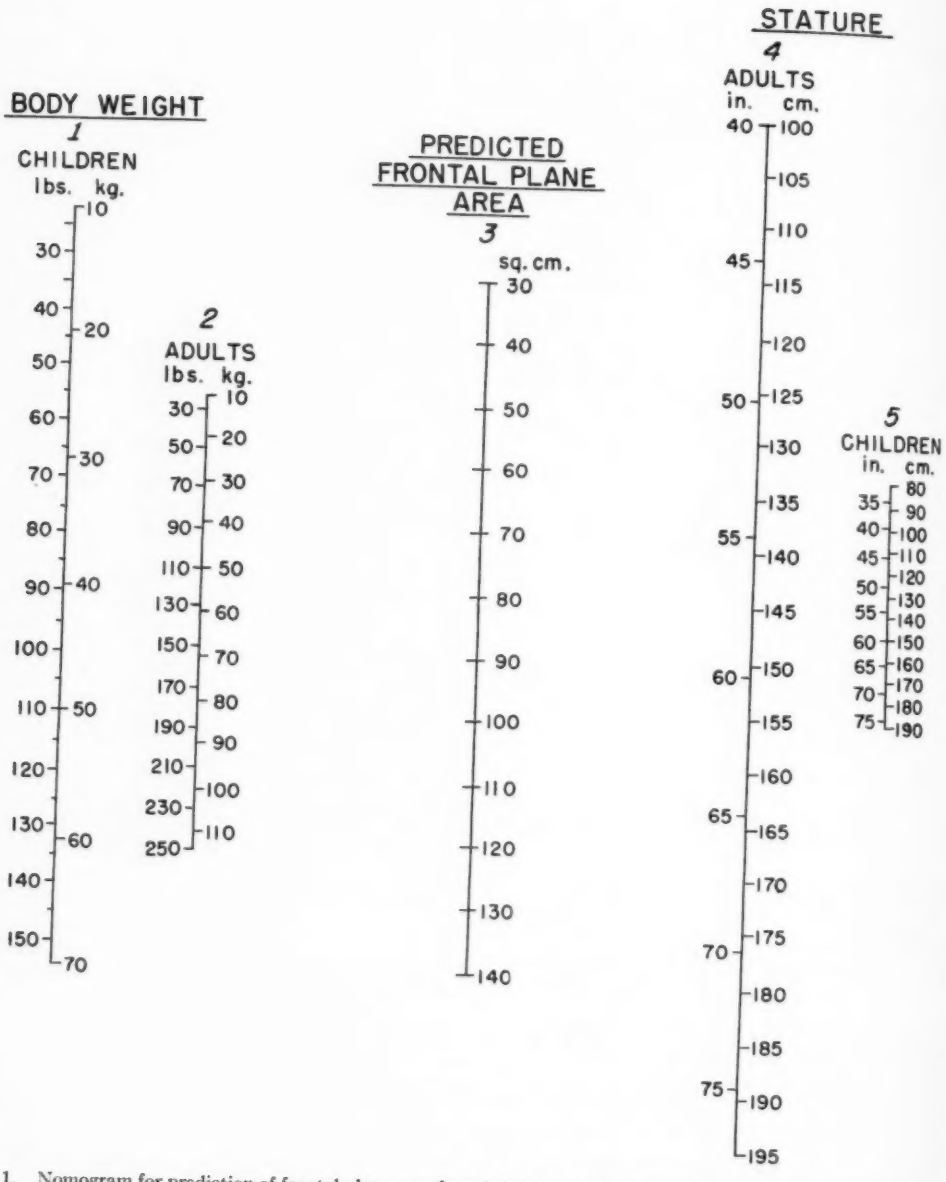

Fig. 1. Nomogram for prediction of frontal plane area from height and weight, according to Equations 2 and 3.
1. For subjects 17 years old, use children scales (1 and 5) unless stature exceeds 170 cm., in which case use adult scales.
2. Indicate weight and stature on respective scales. Lay a ruler on these points and at its intersection with Scale 3 read the predicted frontal plane area of the heart.

from approximately 5 cm. to 28 cm. depending upon the patient).

This equation assumes that the patient faces the film, and the final term $C/3D$ depends upon Bardeen's demonstration that the frontal plane of the heart lies approximately one-third of the way back along the anteroposterior axis of the chest.

If the patient stands back to film, the final term becomes $2C/3D$. Almost all of our measurements are done with the patient facing the film; D approximately 6 feet; K, the inherent distance between the film in the cassette changer and the bakelite sheet against which the patient stands, 3 cm. Sometimes, however, it is convenient to be able to attempt measurements under less satisfactory conditions (for example, with the patient standing back to film, in which case the final term becomes $2C/3D$), and for various values of D and K.

Since the values of K and D will usually be fixed for a particular machine, it is a simple matter to reduce Equation 1 to a graph from which the factor F can be read as a function of anteroposterior chest diameter (C), or factor F may be included as one of the columns in an alignment chart or nomogram.

PREDICTION OF NORMAL AREA

For adults ([1]) the formula is

$$\text{Frontal plane area} = 0.87H + 0.34W - 63.8 \quad (2)$$

where H = subject's height in cm. and W = weight in kg. For children ([2]) the formula is

$$\text{Frontal plane area} = 0.180H + 1.045W + 13.7 \quad (3)$$

NOMOGRAMS

The completion of the upper and lower borders of the silhouette requires some experience but takes only a few seconds of time, and the paper tracing is quickly and easily made. The computations would be far too tedious if they had to be done for each examination, but by reducing the various equations to alignment charts or nomograms (Figs. 1 and 2) all computations are avoided. There remains the matter of measuring the area of the silhouette with a planimeter which, it must be admitted, is a rather exacting and time-consuming procedure.

SUBSTITUTION OF PRODUCT OF LONG AND SHORT DIAMETERS FOR AREA OF THE SILHOUETTE

At a meeting of U. S. Public Health consultants at Bethesda, Md., in the fall of 1944, I reported work that Dr. Gerhart Schwarz of our staff had been doing on heart measurements in 4 × 5-inch photoroentgenograms as a precaution against the possibility that the wartime film shortage might force us temporarily to abandon the use of 14×17-inch chest films. In the discussion that followed, Dr. Edward Chamberlain pointed out that certain combinations of diameters, such as the product of long and short diameters, unlike the transverse diameter, correlate closely with area, not only in the normal but also in the pathologically enlarged heart. Since planimeter measurements are particularly troublesome in the case of small films, he suggested that we might find it worth while to substitute for them the product of the long and short diameters. The long diameter of the silhouette is a straight line arising at the cardiovascular junction at the upper right aspect of the silhouette and extending to the apex of the heart at the lower left aspect. The short diameter is the sum of two segments: A, the perpendicular distance from the long diameter to the most distant portion of the right border, plus B, the perpendicular distance from the long diameter to the left border.

Following up this suggestion, Dr. Schwarz has studied the publications to which Dr. Chamberlain referred and has derived his own equation. He concludes that the best way to determine the size of the frontal plane area is to measure it with a planimeter, as we have been doing for years, but that without much sacrifice of accuracy one may simplify the technic by measuring the long and short diameters and multiplying their product by the factor 0.735.

In the case of miniature films, the area obtained will be that of the reduced size

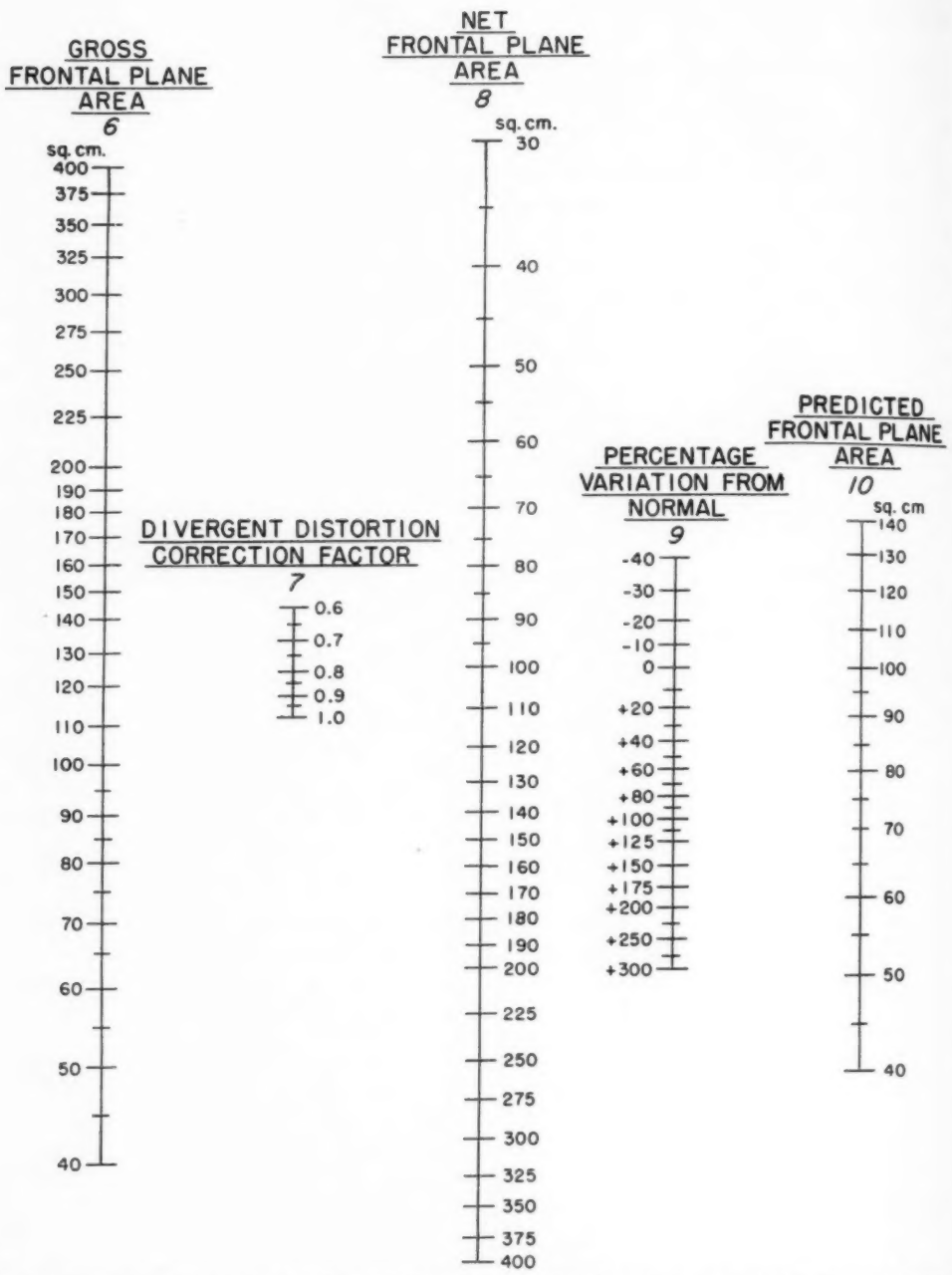

Fig. 2. Nomogram for correcting for divergent distortion and estimating percentage variation of measured area from predicted area.

1. Indicate on Scale 10 the predicted frontal plane area obtained from Scale 3 of Fig. 1.
2. Measure the tracing of the silhouette with a planimeter and indicate the measured value on Scale 6.
3. From appropriate graphs of Equation 1 and a knowledge of the AP diameter of the patient's chest, determine divergent distortion correction factor and indicate it on Scale 7.
4. Lay a ruler on the indicated points on Scales 6 and 7 and at its intersection with Scale 8 indicate the net frontal plane area.
5. Lay a ruler on the indicated points on Scales 8 and 10 and at its intersection with Scale 9 read and record the percentage by which the heart exceeds or is less than the normal predicted area for the subject being examined.

image, which in turn must be multiplied by the square of the linear minification factor, M^2, in order to obtain gross frontal plane area. This in turn must be multiplied by the divergent distortion correction factor in order to obtain net frontal plane area. This procedure sounds complicated but, as a matter of fact, can be rendered simple by the construction of suitable alignment charts.

EVALUATION OF RESULTS

We expect to substitute Dr. Schwarz's Equation 4 (3) for planimetry in our heart measurements from 14×17-inch chest films, believing that the saving of time more than offsets the slight reduction in accuracy; and if the time were to come when stereoscopic 4×5-inch films were used by us as substitutes for 14×17's, rather than as a mere preliminary screen, we would employ Schwarz's Equation 6 with them. At present, however, like most others, we use 4×5-inch filming merely as a screen, and 35-mm. and 70-mm. filming probably always will be used exclusively for screening. If the cost of small film screening is to be held to a minimum, the films should be non-stereoscopic, a phototimer should make the exposure automatically, the accompanying record should be reduced to essentials, such as name and identifying number, and the subsequent reporting should amount to nothing more than a sorting into normals and non-normals, the latter to be returned for large film raying and eventual diagnosis. Clearly, quantitative heart measurement has no routine place in such a scheme, but perhaps Dr. Schwarz's nomograms can be used as the basis from which to develop a coarse, rapidly applied criterion which, regardless of height, weight and anteroposterior chest diameter, will select those subjects whose hearts are so clearly enlarged as to merit return for large film study.

Division of Roentgenology
University of Chicago
Chicago 37, Ill.

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Determination of Frontal Plane Area from the Product of the Long and Short Diameters of the Cardiac Silhouette[1]

GERHART S. SCHWARZ, M.D.

The Division of Roentgenology, The University of Chicago

THE PRECEDING article, by Dr. Paul C. Hodges([4]), describes the events leading up to the work to be reported here on the correlation between frontal plane area and the long and short diameters of the cardiac silhouette. For convenience two equations listed in that paper are reproduced:

$$F = \left[1 - \frac{K}{D} - \frac{C}{3D}\right]^2 \qquad (1)$$

where F = divergent distortion correction factor; K = distance in cm. between anterior surface of chest and film (in our apparatus, 2.5 cm.); D = distance in cm. between target of x-ray tube and surface of film (in our apparatus, 183 cm.); C = anteroposterior diameter of chest in cm. (varies from approximately 5 cm. to 28 cm., depending upon the patient).

$$A = 0.87H + 0.34W - 63.8 \qquad (2)$$

where A = frontal plane area of heart in sq. cm.; H = subject's height in cm.; W = subject's weight in kg.

The material studied consists of 99 teleroentgenograms made on 93 subjects examined at the University of Chicago. Group 1 consists of 25 films of normal subjects (10 males, 15 females) of ages varying from 7 to 54 years, weight from 18 to 118 kg., height from 132 to 177.5 cm., and cardiac area from 72 to 145.5 sq. cm. Group 2 consists of 74 films made on 68 subjects in whom clinical or x-ray evidence indicated heart disease. Thirty-five were males, 33 females; the ages varied from 6 to 82 years, weight from 14.1 to 88.9 kg., height from 100 to 185 cm., and cardiac area from 50 to 282 sq. cm. Area ranged from 32 per cent undersize to 110 per cent oversize for build as computed by the Hodges and Eyster formula (Equation 2). In Group 2, three subjects were examined twice, one subject four times, during the course of cardiac disease.

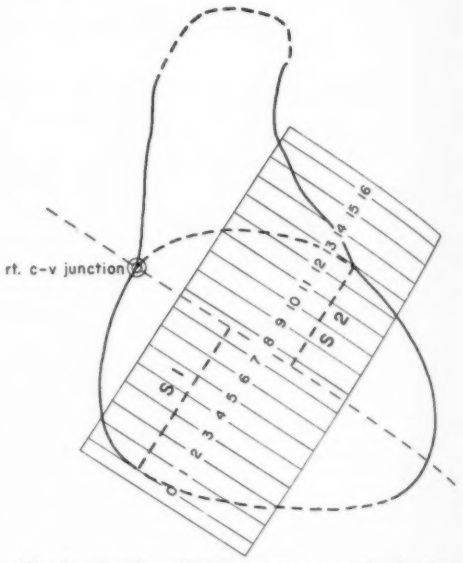

Fig. 1. Tracing of teleroentgenogram showing frontal plane area, long and short diameters of silhouette and transparent ruler for measuring short diameter.

The long diameter, L, extends from the cardiovascular junction, upper right, to the cardiac apex, lower left. The short diameter, S, is most easily measured with the aid of a superimposed transparent ruler. With such a ruler aligned so that its lines are parallel with L and its zero line is tangent to the most prominent part on the lower border of the silhouette, one reads S as the most prominent point on the upper border of the silhouette. If such a special ruler is not available, it may be necessary to measure $S1$ and $S2$ separately and then add these values to obtain S.

The films were made with the subject standing or sitting facing a vertical cassette changer and with a target-film distance (D) of approximately 6 feet. In most instances, filming was stereoscopic

[1] Read by title at the Thirty-first Annual Meeting of the Radiological Society of North America, Chicago, Ill., Nov. 9–10, 1945.

and the tracing was made from the larger cardiac silhouette in the stereoscopic pair.

WORK-UP OF FILMS AND TRACINGS

The silhouettes were outlined with wax pencil on the film and then transferred to semitransparent paper for planimetric measurement of area and linear measurement of the long and short diameters. This latter measurement may be done with an ordinary ruler but is made easier with the aid of a special rectangular transparent ruler (Fig. 1). For each case the product of these diameters was divided into the area of the silhouette, the quotient being a factor which is indicated as F in Column 11 of Tables I and II.

TABULATION

Table I lists the values for the normals; Table II, for subjects with abnormal hearts. There are 13 columns in each table. Column 1 lists the x-ray number; 2, the sex; 3, the age; 4, the height in cm.; 5, the weight in kg.; and 6, the anteroposterior diameter of the chest in cm. Column 7 shows the frontal plane area in sq. cm. (A_m) as measured by the planimeter; Column 8, the long diameter (L) of the silhouette in cm.; Column 9, the short diameter (S) of the silhouette in cm. Column 10 lists the product of L and S; Column 11, the factor F, which is the quotient obtained by dividing the value in Column 7 by the value in Column 10. Column 12 lists the area (A_e) obtained by computation from the formula for the ellipse ($L \times S \times 0.785$); Column 13 lists area ($A_{0.735}$) computed by the 0.735 formula presently to be described.

COMPUTATION

For the normals the mean value of F was 0.740; for the abnormals 0.730; the medial 0.735 was therefore chosen as the factor for general application. On this basis, the equation for the calculation of frontal plane area from long and short diameter becomes

$$A = L \times S \times 0.735 \qquad (4)$$

where A = gross frontal plane area of the heart in sq. cm. in full-sized roentgenograms made with the patient facing the film at a target-film distance of approximately 72 inches; L = long diameter of cardiac silhouette in cm.; S = short diameter of cardiac silhouette in cm.

From other material studied, it appears that the factor depends to a minor degree upon target-film distance (D) and to a somewhat greater degree on the relationship of the chest to the film. When the patient is back to film, the factor becomes considerably smaller and when D is increased beyond 72 inches it becomes somewhat larger, being maximum for orthodiascopy where, of course, D may be considered as being infinitely great.

SIMILAR WORK BY OTHERS

Moritz[10] in 1931 divided the product of long and short diameters into frontal plane area as determined by orthodiascopy and obtained a factor of 0.763 when the patient was in the horizontal position and 0.752 when the patient was erect.

Ludwig[8] in 1939, also using orthodiagrams and working on 120 normal Swiss soldiers, obtained a factor of 0.725. A number of workers[5, 6, 15], including two insurance company examiners[13], used the factor 0.785 on the assumption that the frontal plane area is an ellipse.

ACCURACY OF FORMULAE

The material in Columns 12 and 13 of the two tables was collected to allow testing the relative validity of Equation 4 and the formula for the ellipse as predictors of frontal plane area. Inspection of Columns 7, 12, and 13 shows that in most instances the predictions by the ellipse formula are larger than the actual measured area, whereas the predictions by the 0.735 formula are sometimes a little larger, sometimes a little smaller, than the actual measured area. The difference is brought out graphically in Figure 2, where the predictions from the ellipse formula, plotted as circles, lie for the most part above the axis; whereas the predictions from the

TABLE I: NORMAL HEARTS: DATA FROM 25 FILMS ON 25 SUBJECTS

1 X-Ray No.	2 Sex	3 Age	4 Height, cm.	5 Weight, kg.	6 AP Chest Diameter, cm.	7 A_m	8 L	9 S	10 $L \times S$	11 F	12 A_e	13 $A_{0.735}$
117,018	F	35	151.0	118.0	27.0	102.0	13.3	10.1	134.33	0.7593	105.5	98.5
117,038	M	8	147.0	18.0	14.0	72.0	10.8	9.3	100.44	0.7163	78.1	74.0
117,037	M	27	177.0	73.6	21.0	136.5	15.5	11.9	184.45	0.7400	145.5	135.8
117,030	M	7	132.0	43.0	16.0	70.6	10.7	9.2	98.44	0.7171	77.0	72.0
117,029	F	26	147.0	47.6	16.5	88.2	12.1	10.1	122.21	0.7217	96.0	89.9
117,024	M	28	177.5	76.5	22.0	145.5	15.0	13.3	199.50	0.7293	156.6	146.5
117,041	F	31	167.0	64.3	20.5	100.0	13.3	10.5	139.65	0.7160	109.8	102.5
116,990	F	21	166.0	59.0	21.0	99.5	12.7	10.8	137.16	0.7254	107.8	100.7
116,991	F	34	157.5	58.2	21.0	111.5	13.9	10.8	150.12	0.7427	118.0	110.8
116,985	F	53	160.0	48.0	21.0	112.5	14.0	10.9	152.60	0.7372	120.0	112.5
116,981	F	35	169.5	68.4	21.5	111.5	14.1	11.1	156.51	0.7124	123.0	115.3
116,982	M	32	181.0	81.0	23.5	135.0	14.8	12.5	185.00	0.7297	145.0	136.2
116,980	F	24	164.0	130.0	20.5	108.3	13.5	10.9	147.15	0.7359	115.5	108.0
116,979	M	13	164.0	51.5	15.0	91.5	12.2	10.3	125.66	0.7282	98.7	89.2
116,975	M	21	178.0	46.0	19.0	124.5	14.0	12.2	170.80	0.7289	134.0	125.9
116,974	F	36	157.5	67.3	21.0	92.2	13.2	9.6	126.72	0.7275	99.5	93.0
117,005	M	20	174.0	63.6	21.0	105.5	13.6	10.7	145.52	0.7249	113.8	106.3
117,001*	M	7.5	136.0	29.4	14.0	78.0	11.4	9.5	108.30	0.7202	85.0	79.7
116,996	F	28	172.0	56.8	19.0	109.0	13.8	10.6	146.28	0.7451	115.0	107.2
116,997	F	27	155.0	49.5	18.0	96.0	12.7	10.3	130.81	0.7339	102.6	95.9
116,998	F	52	157.0	63.6	25.0	106.0	14.0	10.5	147.00	0.7210	115.5	107.7
116,999	F	17	157.0	59.0	19.0	92.0	12.3	10.2	125.46	0.7333	98.5	92.1
116,994	F	24	167.0	60.5	19.5	132.6	14.9	12.3	183.27	0.7235	143.5	134.8
116,995	F	22	160.0	66.0	20.0	115.3	13.8	11.8	162.84	0.7081	128.0	120.0
116,993	M	54	167.5	58.6	21.0	117.3	13.6	11.5	156.40	0.7500	122.5	115.0

* Subject sitting rather than standing.
A_m = Gross frontal plane area in sq. cm. as measured by planimeter.
A_e = Frontal plane area in sq. cm. calculated by ellipse formula.
$A_{0.735}$ = Frontal plane area in sq. cm. calculated by 0.735 formula.

L = Long diameter of cardiac silhouette in cm.
S = short diameter of cardiac silhouette in cm.
F = factor obtained by dividing A_m by $L \times S$.

COMPUTED FRONTAL PLANE AREA IN SQ. CM.

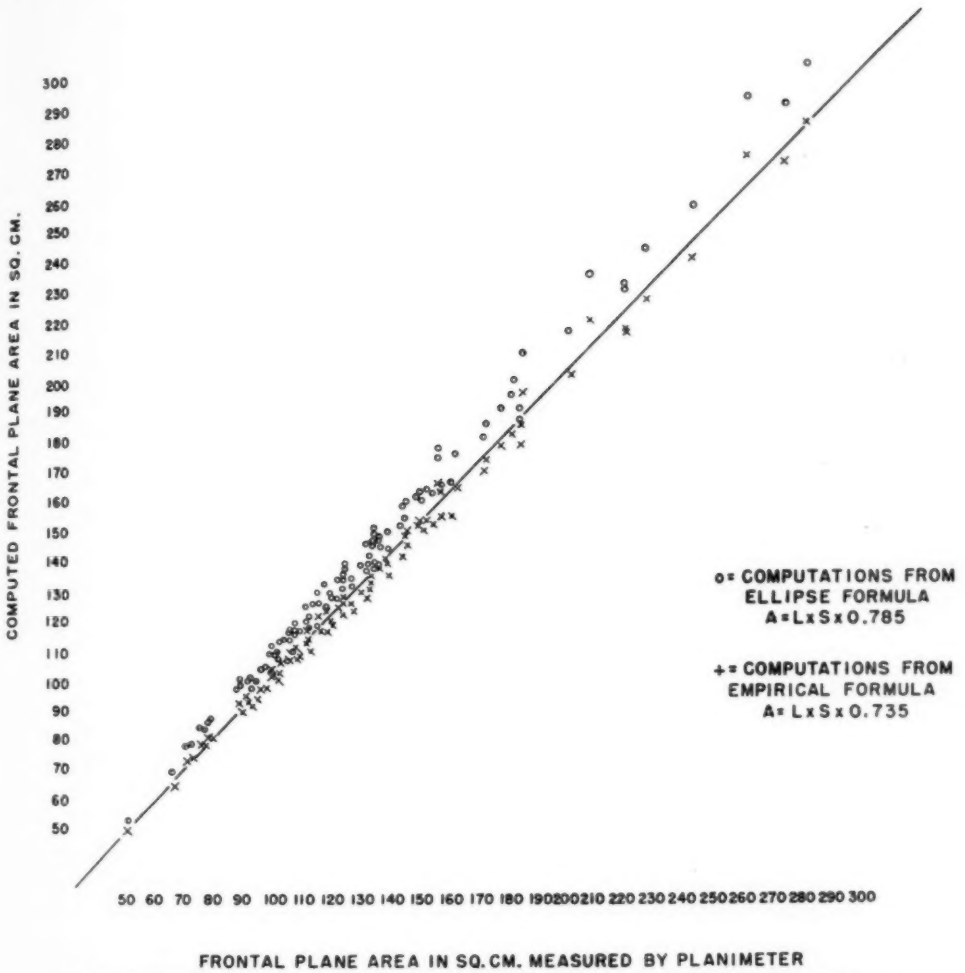

Fig. 2. Relative accuracy of 0.735 formula and ellipse formula in computing frontal plane area. Values computed from Equation 4 ($A = L \times S \times 0.735$), indicated as +, are distributed evenly about the axis, while those computed from the ellipse formula ($A = L \times S \times 0.7854$), indicated as 0, tend to be too large.

0.735 formula, represented as crosses, are arranged symmetrically on both sides of the line of regression. The distribution of errors has been tabulated as follows:

Formula for the Ellipse: The deviation of calculated values from measured values was as follows:

	Cases
1% or less	1
More than +1% to 2%, inclusive	2
More than +2% to 3%, inclusive	4
More than +3% to 4%, inclusive	9
More than +4% to 5%, inclusive	13
More than +5% to 12%, inclusive	70

The maximum deviation was +11.83 per cent or +31 sq. cm. In only one instance was the deviation zero. A negative deviation did not occur.

Formula $L \times S \times 0.735$: The deviation of calculated values from measured values was as follows:

	Cases
±1% or less	30
More than ±1% to ±2%, inclusive	32
More than ±2% to ±3%, inclusive	15
More than ±3% to ±4%, inclusive	11
More than ±4% to ±5%, inclusive	9
More than ±5% to ±6.5%, inclusive	2

TABLE II: ABNORMAL HEARTS: DATA FROM 74 FILMS ON 68 SUBJECTS

1 X-Ray No.	2 Sex	3 Age	4 Height, cm.	5 Weight, kg.	6 AP Chest Diameter, cm.	7 A_m	8 L	9 S	10 $L \times S$	11 F	12 A_s	13 $A_{0.725}$
119,229	M	39	172.6	79.7	25.5	244.0	20.80	15.60	324.48	75.20	255.0	238.0
111,293	M	31	171.0	67.0	20.5	134.0	15.80	11.60	183.28	73.11	144.0	135.0
117,088	F	61	167.0	52.0	20.0	137.0	15.20	12.00	182.40	75.10	143.0	134.0
122,019	F	44	154.3	54.4	18.0	106.0	13.00	11.00	143.00	74.13	112.0	105.0
122,245	M	18	174.1	61.0	20.5	125.0	14.50	12.00	174.00	71.83	136.5	128.0
122,115	M	20	170.0	79.6	21.0	155.0	16.10	12.70	204.47	75.80	160.5	150.5
122,395	M	52	159.0	56.0	22.0	262.0	20.60	18.00	370.80	70.65	291.0	272.0
122,028	M	64	175.5	64.0	22.0	228.0	19.40	15.80	306.52	74.38	241.0	225.0
121,931	F	37	157.0	58.4	19.5	109.0	13.40	11.10	148.74	73.28	117.0	109.0
94,761	M	5	114.0	19.6	13.0	65.5	10.80	8.10	87.48	74.87	68.7	64.5
38,440*	M	11	145.6	34.9	16.5	75.0	11.70	9.10	106.47	70.44	83.5	78.0
38,440*	M	12	153.9	36.8	17.5	94.5	12.10	10.40	125.84	75.10	98.8	93.0
38,440*	M	12	153.9	36.8	17.5	77.0	11.60	9.10	105.56	72.94	83.0	77.5
38,440*	M	17	173.2	62.3	16.5	103.0	13.70	10.35	141.80	72.64	111.2	104.0
73,621*	F	19	154.0	47.0	20.0	127.5	14.60	11.30	164.98	77.20	129.5	121.5
73,621*	F	19	155.0	42.9	18.5	101.0	13.05	10.50	137.03	73.71	107.5	100.5
77,880	F	37	162.8	66.2	20.5	139.5	15.05	12.45	186.75	74.43	147.0	138.0
77,774*	F	65	162.0	63.2	19.5	127.3	14.20	11.90	168.98	75.33	132.8	124.5
77,774*	F	66	162.0	63.0	19.5	136.5	14.70	12.70	186.69	73.12	146.5	137.5
77,649	F	30	170.0	57.2	19.0	185.0	18.55	12.95	240.22	77.01	188.5	176.5
76,727	M	56	175.5	73.0	19.0	157.3	16.25	13.50	219.38	71.70	172.0	151.5
79,003	M	19	170.0	63.9	19.5	135.0	14.80	12.65	187.22	72.11	147.0	137.5
122,795	M	53	171.0	68.1	21.5	139.0	16.15	11.10	183.15	75.89	148.3	139.0
122,915	F	43	173.0	76.6	22.5	127.0	14.25	11.90	169.58	74.89	133.3	124.5
78,993	M	63	177.0	91.8	27.5	185.0	18.50	12.70	234.95	78.74	185.0	173.0
78,559	M	55	163.0	61.2	22.0	153.0	15.50	13.30	206.15	74.22	162.0	151.5
79,594	M	64	172.7	87.4	24.0	144.0	15.00	12.70	190.50	75.59	150.0	140.0
79,565	M	75	162.2	64.9	24.0	158.0	17.50	11.90	208.25	75.87	163.5	153.0
79,652	F	42	150.0	70.0	25.5	132.0	15.20	11.30	171.76	76.85	135.0	126.5
82,214	F	11	145.0	31.0	16.0	92.8	12.40	9.90	122.76	75.59	96.5	90.0
80,892	M	64	174.2	62.8	22.0	161.5	16.80	12.40	208.32	77.52	164.0	153.0
52,651*	F	20	162.8	62.4	19.0	120.5	13.70	11.70	160.29	75.18	126.0	117.5
52,651*	F	23	162.8	64.3	19.0	124.5	14.30	11.80	168.74	73.78	132.5	124.0
78,490	F	59	154.3	60.0	20.0	133.5	15.10	11.80	178.18	74.92	140.0	131.0
78,542	M	28	171.7	60.8	19.0	163.2	16.50	13.40	221.10	73.81	173.5	162.5
78,042	F	9	137.0	28.3	15.0	104.0	13.10	10.90	142.79	72.83	112.2	105.0
77,939	F	22	159.8	51.2	17.5	116.0	13.70	11.60	158.92	72.99	124.5	116.5
77,898	F	55	156.5	74.9	22.5	130.0	14.30	12.20	174.46	74.52	137.0	128.0
80,785	M	48	185.0	79.6	21.0	172.5	17.60	13.00	228.80	75.39	179.5	168.0
80,695	M	61	176.0	88.9	25.5	173.5	17.80	13.10	233.18	74.41	183.0	171.5

77,898	F	55	156.5	74.9	22.5	130.0	14.30	12.20	174.46	74.52	137.0	128.0
80,785	M	48	185.0	79.6	21.0	172.5	17.60	13.00	228.80	75.39	179.5	168.0
80,695	M	61	176.0	88.9	25.5	173.5	17.80	13.10	233.18	74.41	183.0	171.5
80,449	F	62	167.5	56.6	19.5	133.0	14.50	12.10	175.45	75.81	137.5	129.0
79,986	F	82	156.0	55.0	18.0	124.0	14.90	11.00	163.90	75.66	128.5	120.5
79,711	M	59	164.6	79.7	26.0	146.0	15.60	12.90	201.24	72.55	158.0	148.0
79,656	F	19	150.0	59.1	20.0	130.5	14.20	12.30	174.66	74.72	137.0	128.5
123,933	F	21	159.6	53.2	18.0	102.0	13.30	10.40	138.32	73.74	108.5	101.5
21,616	M	75	167.5	65.0	18.5	122.5	14.70	11.45	168.32	72.78	132.0	122.5
79,479	F	53	147.5	51.8	22.0	145.5	16.05	12.10	194.21	74.92	152.5	143.0
80,625	M	39	171.4	54.4	18.5	108.0	13.60	11.00	149.60	72.19	117.5	109.5
78,027	M	26	178.5	53.4	21.0	98.0	13.05	10.10	131.81	74.35	103.5	96.5
77,682	M	58	174.2	66.0	22.0	114.0	13.90	11.35	157.77	72.26	124.0	116.0
77,587	F	21	175.3	62.9	18.5	79.0	11.50	9.50	109.25	72.31	86.6	80.0
83,027	F	24	172.1	70.0	20.5	140.0	15.25	11.90	181.48	77.14	142.5	133.5
82,864	F	26	160.0	68.6	20.0	135.0	15.30	12.40	189.72	71.16	149.0	139.5
88,718	F	47	153.0	46.5	21.0	157.0	16.60	13.40	222.44	70.58	175.0	163.5
88,146	F	28	158.1	69.1	24.5	125.0	14.85	11.80	175.23	71.33	137.5	129.0
88,143	M	58	175.0	49.3	21.5	186.0	19.20	13.75	264.00	70.45	207.0	194.0
87,606	F	23	153.1	54.5	20.5	113.0	13.70	10.80	147.96	76.37	116.2	108.5
86,058	F	39	150.5	56.3	21.5	118.5	14.50	10.80	156.60	75.67	123.0	115.0
85,638	F	25	144.0	68.0	29.0	133.5	14.90	12.20	181.78	73.44	143.0	133.5
82,629	F	46	162.2	54.0	19.0	108.0	13.30	10.90	144.97	74.50	114.0	106.5
99,227	M	27	180.0	81.8	21.5	151.0	15.80	13.00	205.40	73.52	161.0	151.0
98,523	M	44	185.0	94.1	24.0	150.0	15.70	13.00	204.10	73.49	160.0	150.0
53,777	F	21	158.1	53.6	18.0	118.0	14.10	11.80	166.38	70.92	130.5	122.0
124,758	F	6	100.0	16.4	13.0	50.0	9.40	7.10	66.74	74.92	52.4	49.3
122,442	M	30	163.0	55.4	19.0	151.0	16.70	12.10	202.07	74.73	158.5	149.0
121,459†	M	70	166.0	70.8	23.0	221.0	20.00	14.60	292.00	75.68	229.5	214.5
116,956	M	57	186.0	75.4	27.0	275.0	21.40	17.20	368.08	74.71	289.0	270.5
118,601	M	48	177.7	86.2	26.0	282.0	22.00	17.50	385.00	73.25	302.0	283.0
119,288	F	60	150.0	70.0	24.0	182.0	18.90	13.00	245.70	74.07	193.0	180.0
117,334	M	6	110.0	14.1	12.0	89.0	12.60	9.80	123.48	72.08	97.0	90.5
120,189	M	64	172.5	71.5	22.0	221.0	20.50	14.20	291.10	75.91	228.5	214.0
120,433	M	36	166.0	62.0	21.0	202.0	19.20	14.20	272.64	74.09	214.0	200.0
122,709	M	47	171.0	90.5	27.5	209.0	19.40	14.90	289.06	72.30	233.0	218.0
120,386	F	23	162.0	56.9	19.5	178.5	17.90	13.40	239.86	74.42	188.5	176.0

* Films taken at intervals during course of disease.
† Subject sitting rather than standing.
A_m = Gross frontal plane area in sq. cm. as measured by planimeter.
A_e = Frontal plane area in sq. cm. calculated by ellipse formula.
$A_{0.735}$ = Frontal plane area in sq. cm. calculated by 0.735 formula.
L = Long diameter of cardiac silhouette in cm.
S = Short diameter of cardiac silhouette in cm.
F = Factor obtained by dividing A_m by $L \times S$.

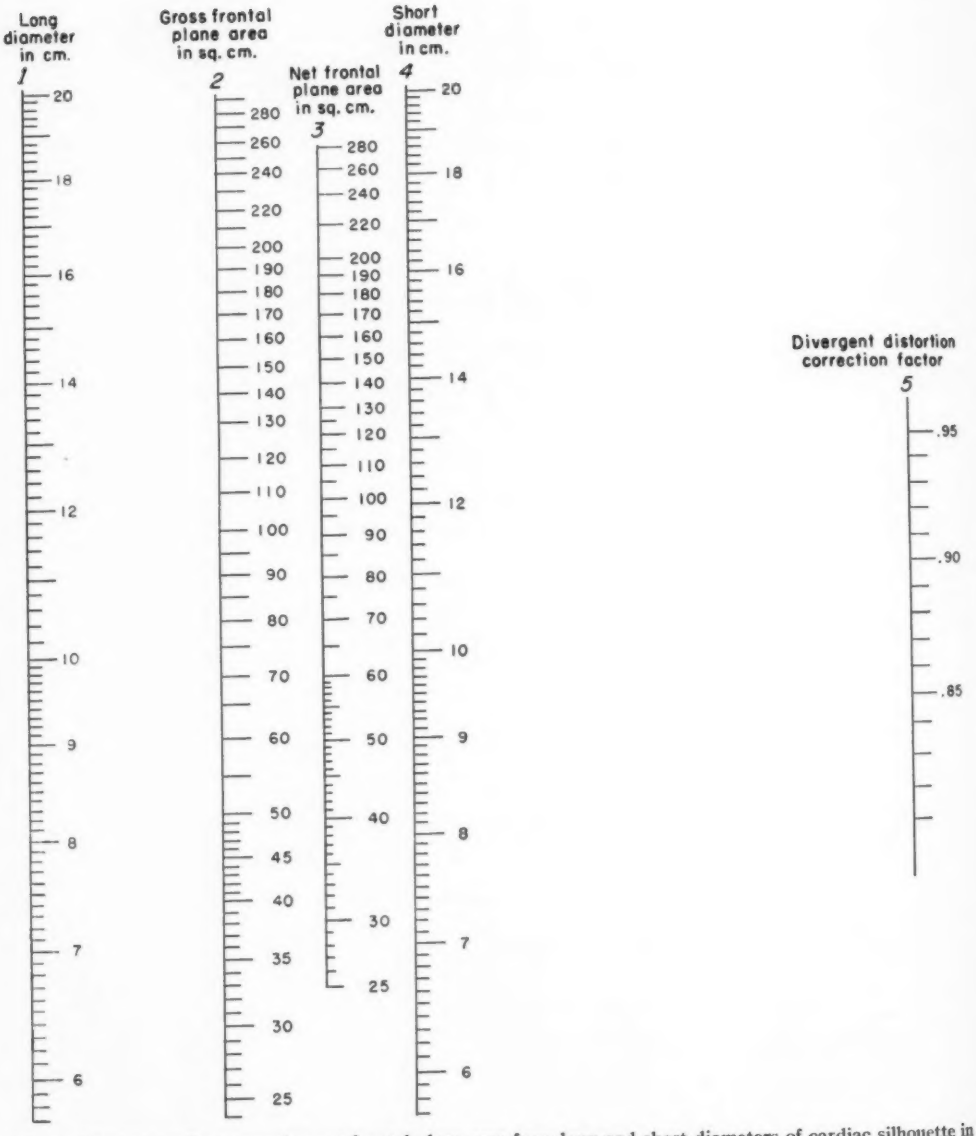

Fig. 3. Nomogram for computing net frontal plane area from long and short diameters of cardiac silhouette in full-sized teleroentgenograms.

1. Measure long and short diameters and indicate these values on Scales 1 and 4.
2. Place a ruler on the indicated points on Scales 1 and 4 and indicate on Scale 2 the gross frontal plane area.
3. By means of a graph or alignment chart of Equation 1, determine divergent distortion correction factor and indicate its value on Scale 5.
4. Place a ruler on the indicated points on Scales 2 and 5 and read on Scale 3 the net frontal plane area of the cardiac silhouette.

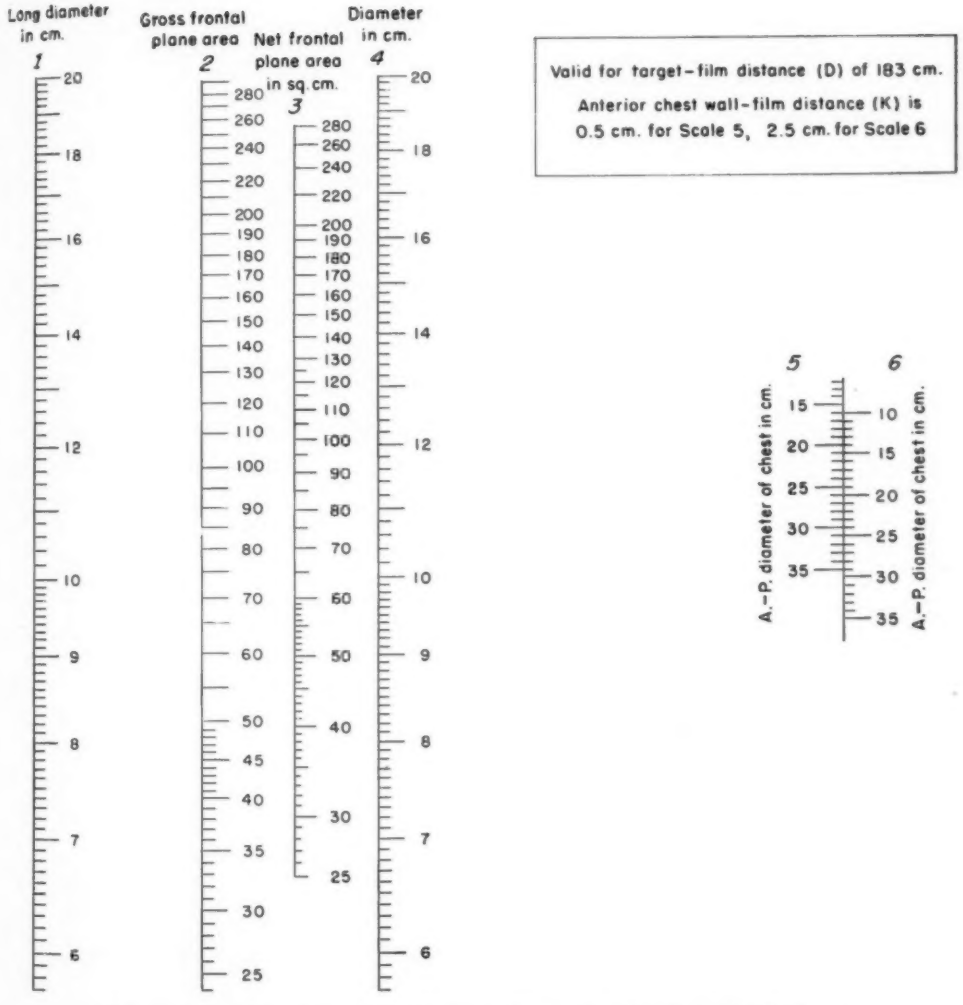
Long diameter in cm.
1
Gross frontal plane area
2
Net frontal plane area in sq. cm.
3
Diameter in cm.
4
Valid for target-film distance (D) of 183 cm.
Anterior chest wall-film distance (K) is
0.5 cm. for Scale 5, 2.5 cm. for Scale 6
5
6
A.-P. diameter of chest in cm.
A.-P. diameter of chest in cm.

Fig. 4. Same as Fig. 3 but with addition of alignment chart for divergent distortion.

1. Measure long and short diameters and indicate these values on Scales 1 and 4.
2. Place a ruler on the indicated points on Scales 1 and 4 and indicate on Scale 2 the gross frontal plane area.
3. Measure patient's AP chest diameter in cm. and indicate this value on Scale 5 if the chest is in direct contact with the cassette or on Scale 6 if the film is in the stereoscopic changer.
4. Place a ruler on the indicated points on Scale 2 and on Scale 5 or 6 and then read on Scale 3 the net frontal plane area.

Note: While the chart is designed for a D of 183 cm. and a K of 0.5 cm. (Scale 5) or 2.5 cm. (Scale 6), the prediction will be only 3 per cent too large if D is as short as 5 feet, and minor variations in K do not introduce serious errors.

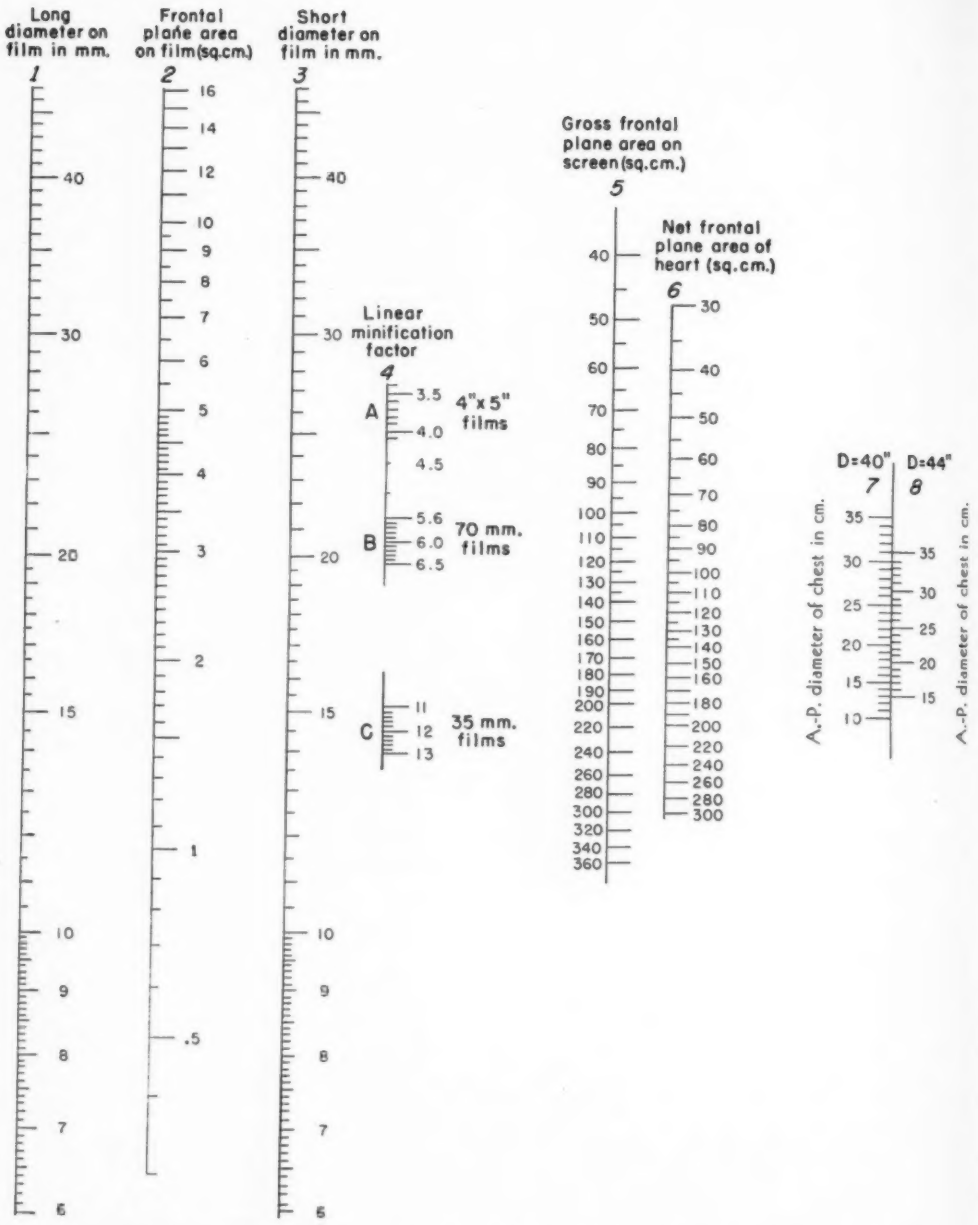

Fig. 5. Nomogram for computing net frontal plane area of cardiac silhouette from measurement of long and short diameters in miniature films.

1. Indicate on the appropriate portion of Scale 4 the linear minification factor of the machine being used.
2. Indicate on Scales 1 and 3 the measured values of the long and short diameters of the miniature silhouette.
3. Place a ruler on the indicated points on Scales 1 and 3 and at the intersection of Scale 2 record the area of the miniature silhouette.
4. Place a ruler on the indicated points on Scales 2 and 4 and at the intersection with Scale 5 indicate gross frontal plane area of the heart (full size).
5. Measure patient's AP chest diameter in cm. and indicate value on Scale 7 if *D* is 40 inches, or on Scale 8 if *D* is 44 inches.
6. Place a ruler on the indicated points on Scale 5 and on Scale 7 or 8 and at the intersection with Scale 6 read the net frontal plane area of the heart.
7. From Fig. 2 of Hodges' paper (page 358) obtain percentage variation from normal.

The maximum deviation was +4.7 per cent and −6.5 per cent, the minimum deviation zero. This indicates that the formula $L \times S \times 0.735$ is more accurate than the ellipse formula.

The accuracy of both formulae has also been compared by statistical methods. The results in abbreviated form are as follows:

Formula for the ellipse: Mean difference between calculated and measured area: +6.49 per cent with a standard deviation of 2.55. Coefficient of correlation: 0.9918.

Formula $L \times S \times 0.735$: Mean difference between measured and calculated area: −0.15 per cent with a standard deviation of 2.39. Coefficient of correlation: 0.9958.

The means of both formulae were compared in order to see whether the discrepancy between them might be caused merely by scattering of values. This was done by comparing the difference between the means of the two formulae with the standard error of this difference. If this difference is greater than 3 times its standard error, it can be said to be statistically significant, since the law of probability indicates that with such a difference there are 99.7 chances out of 100 that the difference is not due to chance. In this case the difference between the means of the formulae is 6.35 ± 0.3495, which shows clearly that it is significant, since it is 18 times greater than its standard error.

MINIATURE FILMS

Tracings of miniature films are made directly on highly transparent paper by means of a sharp-pointed hard lead pencil. The factor of linear minification, M, is obtained from

$$M = \frac{I}{i} \qquad (5)$$

where I = length in cm. of an image on the fluorescent screen and i = length in cm. of the corresponding microfilm image.

The linear minification factor for a particular apparatus may be measured as follows:

A strip of lead pierced with two small holes spaced 10 cm. apart is attached to the front surface of the hood. Target-screen distance (D) and object-screen distance (d) are measured. In the image (I) that will be developed on the fluorescent screen, the distance between the holes will be 10 cm. $\times$ ($D/D-d$). Now a microfilm is made and i is measured on the film. By substituting in Equation 5 the computed value of I and the measured value of i, the linear minification factor is obtained.

In the case of miniature films, the equation for computing gross frontal plane area, A, becomes

$$A = L \times S \times 0.735 \times M^2 \qquad (6)$$

where M is the factor of linear minification.

NOMOGRAMS

Three nomograms are provided to facilitate application of the various formulae, and the bibliography lists several texts that explain the construction of alignment charts.

SUMMARY

1. In the working up of teleroentgenograms, it is possible to avoid use of the planimeter and compute frontal plane area from measurement of the long and the short diameters of the cardiac silhouette.

2. The equation for this computation is:

$$A = L \times S \times 0.735$$

where A = gross frontal plane area in sq. cm.; L = gross long diameter in cm.; S = gross short diameter in cm. It is based on measurement of abnormal as well as normal hearts, and correlation between computed area and planigraphically measured area is high ($r = 0.9958$).

3. The frontal plane silhouette is only approximately elliptical, and the standard equation for the ellipse ($A = L \times S \times 0.785$) yields areas that almost always are too large.

4. Nomograms provided in this and the preceding paper (Hodges) yield net frontal plane area and also the amount by which this value varies from normal.

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Division of Roentgenology
University of Chicago
Chicago 37, Ill.

Esophagopleural Fistula Complicating Empyema[1]

ARTHUR A. BREWER, M.D.
Alton, Ill.

ESOPHAGOPLEURAL fistula secondary to purulent empyema thoracis is an uncommon condition. Seventeen cases from the literature are reviewed here, in most of which the diagnosis was made when particles of ingested food appeared in material from the draining empyema cavity. The case to be recorded is the first in which the diagnosis of esophagopleural fistula was established by roentgen examination prior to any operative procedure.

REPORT OF CASE[2]

A 4-year-old white girl was admitted to Alton Memorial Hospital, Oct. 10, 1944, because of diarrhea and vomiting of one week duration. She had been chronically ill during the past two years, but all treatment had been at home until the present admission. Two years earlier she had had an acute illness and the parents were told she had pneumonia followed by a lung abscess. She apparently recovered in about three weeks but since that time she had had an intermittent chronic cough and frequent colds.

One year preceding admission, depression of the right chest was noticed, and this had become progressively more pronounced. The parents thought that during the three months prior to the present illness the child's health had been better than at any other time in the past two years. During the past year, however, there had been a marked loss of weight and strength. One week before admission there was sudden vomiting of watery, greenish yellow material. Diarrhea and fever followed, and the patient became acutely ill. There was frequent cough, which often stimulated vomiting.

General Examination: The patient was obviously ill. She was emaciated and extremely poorly developed. Her weight was only 19 pounds, and the body appeared to be that of a 2-year-old child. The right chest was flattened at the apex, there was pronounced scoliosis, and the anteroposterior diameter was greatly decreased on the right. There was no respiratory excursion. On the right, breath sounds were diminished; on the left, a few sticky râles were heard. The liver was palpable. The fingers showed early clubbing.

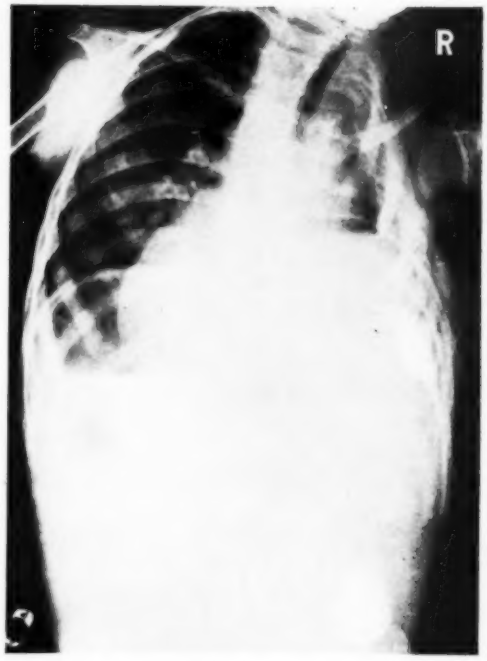

Fig. 1. Admission roentgenogram, showing incomplete development of the right thorax and an opacity over most of the right lung field, with a fluid level and air in the midportion. (The trachea is well visualized and is shifted to the involved side, and should not be confused with free air in the pleural cavity.) Opaque material in the colon and in the lower portion of the right lung field is presumably a medicament, and suggests a communication between the gastro-intestinal tract and the right pleural cavity.

Temperature on admission was 103°. The urine showed 3+ albumin and an occasional hyaline cast. There were 23,800 leukocytes, with 83 per cent neutrophils and 17 per cent lymphocytes. The erythrocyte count was 4,400,000, with 62 per cent hemoglobin.

There was repeated vomiting of foul-smelling green fluid. At the Alton Memorial Hospital the patient was given supportive treatment only.

Roentgen Examination: A postero-anterior view of the chest (Fig. 1), made the day after admission, revealed a complete opacity over most of the right

[1] From the Department of Radiology, Alton Memorial Hospital, Alton, Ill. Accepted for publication in October 1945.

[2] Patient admitted on the pediatrics service of Dr. Noble D. McCormack.

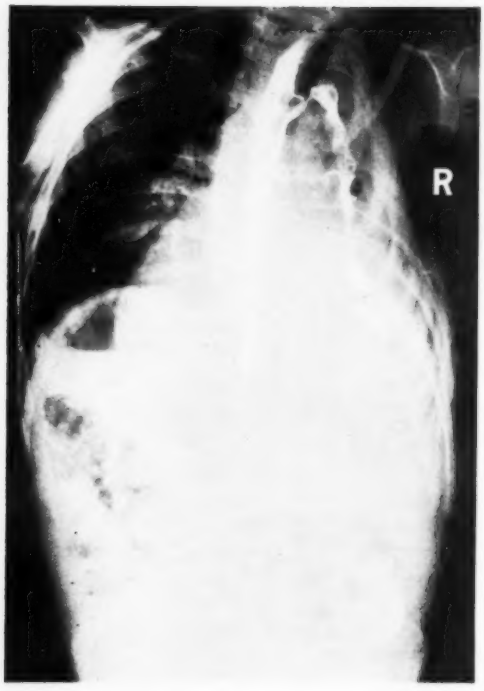

Fig. 2. Esophagogram revealing two clearly defined fistulae from the esophagus to the right pleural cavity.

side, with a fluid level in the mid-portion and air above the fluid. Pronounced lack of development of the right thorax indicated long duration of the condition.

Traces of opaque material seen in the colon were assumed to be some opaque medicament. A faint trace of a substance of similar density was seen in the lower portion of the right chest. This formed a denser shadow within the opacity and apparently was within the pleural effusion. This observation immediately started speculation as to how the opaque material arrived in the pleural cavity. On the assumption that it was similar to that observed in the colon and was actually in the pleural space, the existence of a fistula from the gastro-intestinal tract to the pleural cavity seemed most probable. The history of sudden vomiting of probably purulent material was suggestive of rupture of an empyema into the esophagus.

Fluoroscopic observation of a barium swallow was done, and two fistulae from the esophagus into the empyema cavity were readily visualized. A roentgenogram clearly demonstrated these fistulae at the level of the fifth and seventh dorsal vertebrae (Fig. 2).

Treatment and Course: The patient was referred to the St. Louis Children's Hospital for treatment. On Oct. 21, 1944, Dr. J. Karl Poppe, of the Barnes Hospital surgical chest service, resected segments from the right seventh and eighth ribs. A thoracotomy tube was inserted, draining a very large posterior chronic empyema cavity, containing foul, dark-colored pus, food, and barium. The patient coughed up some of the same material immediately after the incision was made.

On the first postoperative day there was profuse drainage of old barium, pus, and gentian violet, which had been given by mouth. A gastric tube was inserted, through which eight daily feedings were administered. Transfusions and other supportive treatment were given.

On the third postoperative day the patient vomited a tube feeding and some of it appeared on the dressing.

On Nov. 15, 1944, the gastric tube was removed. The patient was given gentian violet by mouth, none of which appeared on the dressing. It was concluded that the fistulae had closed, and subsequently there had been no recurrence.

About two months after operation, ambulatory and afebrile, but with a thoracotomy drainage tube in the right chest, the patient was transferred to Ridge Farm, the St. Louis Children's Hospital convalescent home, where she gained weight and progressed satisfactorily.

In April 1945 she was discharged, completely afebrile and ambulatory, with her thoracotomy entirely closed and no evidence of any residual empyema cavity on roentgen examination. She was last seen in November 1945, thirteen months after the original examination. At that time she was in excellent health, she weighed 35 pounds, and roentgen examination revealed aeration of the involved lung. A barium swallow showed irregularities at the sites of the fistulae but no interference with deglutition.

DISCUSSION

Perforations of the esophagus are not uncommon, and may be due to trauma, including that incident to foreign bodies and instrumentation, to carcinoma, simple ulceration, strong chemicals, rupture of a diverticulum, or spontaneous rupture during severe vomiting. Perforation is usually followed by mediastinitis or empyema, or both. There often result shock, severe substernal pain, dyspnea and cyanosis; death may occur within twenty-four to forty-eight hours. Should the patient survive, esophagopleural fistula with secondary empyema may occur.

Esophagopleural fistula secondary to preexisting empyema is unusual. Blauvelt (5), Torbett and Bennett (17), Kanter

and Madoff (12), and Cohen and Sindell (8) have reviewed the literature within the past ten years. All have included cases due to tuberculous empyema, whereas this review excludes the obviously tuberculous cases. Berman and Walters (4) reported a case of empyema complicated by broncho-esophagopleural fistula; they were unable to find any earlier report of such a fistula, either as a cause or result of empyema.

Data from 17 cases from the literature, and the one here reported, are summarized in Table I. The ages ranged from 2 to 52 years, but only 3 patients were over 17. The cases were approximately equally divided between the sexes, and there was no definite predilection for either side.

Etiology: In this group of cases the fistula was preceded by a purulent empyema. In parts of its course, the esophagus lies in direct contact with the mediastinal pleura. It seems most probable that the empyema creates an inflammation of the esophageal wall, with ulceration, or necrosis, and perforation. The actual rupture of the weakened esophageal wall might occur during the act of swallowing or vomiting.

In some cases, such as the one here reported, the empyema was obviously neglected, but in others there had been prompt and adequate surgical treatment. In McCormick's (13) case a rubber tube was found in the empyema cavity when it was reopened three years after the previous operation.

In 2 of the 7 cases which came to autopsy the fistula communicated with an esophageal diverticulum. It is obvious that a diverticulum, or some other inherent weakness of the esophagus, might be an unproved predisposing factor in any of these cases.

Cohen and Sindell reported 7 cases associated with tuberculous empyema. Tuberculous empyemas are notoriously chronic and there were no cures in these 7 cases. Eleven of the 18 non-tuberculous cases were cured.

Diagnosis has depended chiefly upon observation of particles of food in the drainage from the empyema cavity. Roentgen confirmation has been obtained in some instances.

If the patient is under close observation, the signs and symptoms may be diagnostic; Ballin and Saltzstein (3) recorded a change in physical signs from those of hydrothorax to those of hydropneumothorax. In the presence of hydrothorax, dysphagia may be a premonitory sign. At the time of rupture there is often vomiting of pus, with relief of the dysphagia. This is usually followed by an elevation of temperature and an increase in toxicity as the pleural space is secondarily invaded.

If a thoracostomy or aspiration is done, the presence of food particles in the drainage is diagnostic of communication between the gastro-intestinal tract and the pleural space. If the drainage has the foul sour odor of butyric acid or fermentation, or there is a variegated bacterial flora or yeast cells, such a communication is probable.

The site of the communication can be further localized by instilling an indicator dye, such as gentian violet, into the stomach with a stomach tube. If this dye does not appear in the drainage, it is assumed that the fistula is from the esophagus; if it does appear, such a possibility is not excluded, as the dye might have been regurgitated. Existence of a fistula is confirmed if, after oral administration, the dye appears on the dressing.

The diagnosis can be confirmed by roentgen examination. Such examination is necessary to determine the exact location of the fistula and is the only conclusive diagnostic procedure in the absence of a thoracostomy opening or aspiration.

Treatment of the empyema is the same as in the uncomplicated case; adequate drainage of the empyema cavity usually requires rib resection.

Treatment of the fistula has been chiefly conservative. By tube feeding or gastrostomy the esophagus is put at rest, and the fistula closes spontaneously. Arquellada (2), in 1920, was the first to report

TABLE I: REPORTED CASES OF ESOPHAGOPLEURAL FISTULA COMPLICATING EMPYEMA

Author and Year Reported	Age	Sex	Side	Bacteriology	Duration of Empyema Prior to Fistula	Diagnosis of Fistula	Treatment	Duration of Fistula	Result	Autopsy
1. Samuelson 1894	11	M	R	Pneumococcus	5 years	Food in drainage 1 month after thoracotomy	Thoracotomy	....	Died	Ulcer of esophagus emptied into pleural space
2. Thursfield 1902	4½	M	R	.....	5½ months	Food in drainage 4 days after second rib resection	Two rib resections	2 weeks	Died	Fistula due to erosion by empyema
3. Bullough 1911	16 mo.	F	L	.....	....	Food in drainage first postoperative day; patient had vomited for 1 week	Rib resection	....	Died	Fistula 3 inches above diaphragm, no diverticulum
4. Arquellada 1920	9	M	L	Streptococcus; staphylococcus	2 months	Food in drainage 2 months after rib resection	Rib resection; Levine tube	3 weeks	Cured	....
5. Crawford 1921	17	M	R	.....	1 year+	Food in drainage 1 year after rib resection; roentgen confirmation	Rib resection; gastrostomy	....	Died 3 days after gastrostomy	Diverticulum communicated with pleural cavity
6. Ballin and Saltzstein 1922	15	M	R	Pneumococcus; B. butyricus; yeasts	1 month maximum	Dysphagia relieved by vomiting pus; physical signs changed from those of hydrothorax to those of hydropneumothorax; food in drainage 4 days postoperative; roentgen confirmation	Rib resection; Rehfuss tube for 3 weeks	8 months	Cured	....
7. Fonte 1922	52	M	L	Possibly syphilitic	Months	Food in drainage	Resection; irrigation	Months	Cured	....
8. Hand and Lee 1922	2	M	L	Pneumococcus	5 days	Food in drainage 4 days postoperative; roentgen confirmation	Thoracotomy; tube feeding, then solid food only	6 days (roentgen confirmation of closure at 3 weeks)	Cured	....
9. Vos 1923	8	M	L	Pneumococcus; variegated flora	3 weeks	Food in drainage	Rib resection; stomach tube	3 weeks	Cured	....

[illegible]			R	Pneumococcus	7 years	Food in drainage when	Thoracotomy 4-2-19	....	Died	Diverticulum connecting with fis-

				flora			tube				
10. McCormick 1928	..	..	R	Pneumococcus	7 years	Food in drainage when wound was reopened	Thoracotomy 4-2-19 Resection 5-31-21 Re-opened 8-19-24 at which time a 4½-inch tube was removed; duodenal tube 3 weeks, then gastrostomy	....	Died	Diverticulum connecting with fistulous tract
11. Acuña and Bettinotti 1928	2½	F	R	Variegated flora	....	Food in aspirated material	Rib resection; gastrostomy	....	Died	Yes
12. Mikula 1931	11	..	R	.....	....	Food in aspirated material	Thoracostomy; gastrostomy	5 months	Cured	....
13. Wall 1932	11	M	L	.....	2 weeks	Food in drainage 11 days after rib resection; roentgen confirmation	Rib resection; fluids only by mouth	2 months	Cured	....
14. Caubarrere 1934	8	..	L	.....	....	Food in drainage 6 days after resection; roentgen confirmation	Rib resection	2 months	Cured	....
15. Blauvelt 1938	54	M	R	Staphylococcus aureus	5 weeks	Food in drainage 27 days after rib resection; roentgen and esophagoscopic confirmation	Two rib resections	....	Died	Yes
16. Torbett and Bennett 1941	30	F	L	.....	4 weeks	Food in drainage 1 week postoperative; roentgen confirmation	Drainage tube in empyema cavity; Levine tube	4 weeks	Cured	....
17. Berman and Walters 1943	23 mo.	F	R	Streptococcus; staphylococcus	Few weeks	Food in drainage 4 days after rib resection; roentgen confirmation; 2 months later roentgen diagnosis of broncho-esophagopleural fistula	Rib resection; gastrostomy with tube in duodenum; later surgical closure of fistulae	7 months	Cured	....
18. Brewer 1946	4	F	R	.....	2 years	Roentgen diagnosis prior to operation	Rib resection; stomach tube	3 weeks	Cured	....

tube feeding, and his was the first reported cure.

Blauvelt suggested the possibility of surgically closing the fistula, but such an attempt had not been reported until Berman and Walters successfully cured the first reported broncho-esophagopleural fistula in this manner. Conservative treatment having proved unsuccessful after seven months, they did a two-stage operation, including further rib resection and complete decortication of the empyema cavity. The broncho-esophagopleural fistula was easily identified, the esophagus was dissected loose from the bronchus, and the fistula was closed with interrupted catgut. The bronchus was dealt with in a similar manner.

SUMMARY

1. A case of esophagopleural fistula as a complication of empyema, diagnosed by roentgen examination prior to any operative procedure, is reported.

2. A brief review of similar cases in the literature is given.

209 Professional Bldg.,
Pasadena, Calif.

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The Use of Pyridoxine Hydrochloride in Radiation Sickness[1]

H. L. VAN HALTERN, M.D.

Pontiac, Mich.

FOLLOWING THE publication of Maxfield's results in the treatment of radiation sickness by pyridoxine hydrochloride (vitamin B_6) in 1943 (6), we became interested in this drug and subsequently began its use in radiation sickness at The Henry Ford Hospital. In order to give the drug a thorough trial, it was decided to limit its use to the more severe cases and not to use it prophylactically. With this in view, we arbitrarily classified our cases of radiation sickness into four groups: (1) mild (anorexia and some nausea); (2) moderate (rather severe nausea, distaste for food, and occasional vomiting); (3) severe (pronounced nausea and frequent vomiting); (4) late or inflammatory symptoms due to proctitis, enteritis, and cystitis.

No medication was offered to patients falling in the first group. Those in the other three groups received intravenous injections of 25 to 50 mg. of pyridoxine hydrochloride, in the form of Hexabetalin, beginning with the onset of symptoms. No other medication was given to any patient either prophylactically or otherwise unless specifically indicated. The injections were made at intervals varying from one to four days according to therapeutic response.

The results were arbitrarily classified as: (1) excellent, *i.e.*, complete relief of all symptoms; (2) good, *i.e.*, relief of all vomiting and most of the nausea; (3) poor, *i.e.*, no appreciable abatement of symptoms.

Since inauguration of the use of pyridoxine hydrochloride, 81 patients have received this drug. The age and sex of the patients, the diagnosis, daily total x-ray dosage, and results of medication are given in the accompanying tabulation. Fractional doses to skin tolerance were used in most cases, and the total daily dosage represents the total roentgens, measured in air, given to all fields treated.

From the accompanying table it is seen that, among the total of 81 cases, excellent results were obtained in 44 (54 per cent), and good results in 28 (35 per cent). In 9 cases (11 per cent) vomiting was not entirely relieved and a troublesome degree of nausea remained. Some of the last group had a noticeable relief of symptoms, and others none at all; in general, there was no relief of diarrhea, tenesmus, and urinary frequency, but in many cases nausea and vomiting were diminished. Tincture of opium in a suitable vehicle gave the greatest relief to the Group 4 cases.

Recent literature has added to the possible physiological causes of radiation sickness. Bean, Spies, and Vilter (1) gave x-ray therapy to patients who had a definite vitamin deficiency and who were suffering from various stages of pellagra or peripheral neuritis. They found a known, although rough, correlation between the severity of radiation sickness and the degree of vitamin deficiency. They were also able to show that correction of the vitamin deficiency prior to x-ray therapy was much more efficient in obviating radiation sickness than administration of vitamin B after irradiation was begun. They suggest that the basic disorder in radiation sickness is a disturbance in respiratory enzyme systems.

Jenkinson and Brown (5) have likened the findings to those of shock. They have graphically illustrated the effect of x-rays on the capillary beds, with resultant anoxemia of cells, local capillary dilatation, and finally, loss of plasma into the tissues. They also point out that radiation sickness is most likely to follow irradiation of those

[1] From The Henry Ford Hospital, Detroit, Mich. Accepted for publication in November 1945.

TABLE I: CASES OF RADIATION SICKNESS TREATED BY PYRIDOXINE HYDROCHLORIDE

Case	Age	Sex	Diagnosis	Daily Dose of Radiation	Result of Treatment with Pyridoxine Hydrochloride
1	61	Female	Carcinoma of esophagus	300 r	Excellent
2	37	Female	Sarcoma of chest wall	300 r	Excellent
3	53	Female	Carcinoma of ovary	400 r	Good
4	24	Female	Lymphoblastoma	350 r	Good
5	61	Male	Carcinoma of rectum	400 r	Good
6	61	Male	Carcinoma of bladder	400 r	Poor
7	38	Female	Marie-Strümpell arthritis	400 r	Good
8	58	Female	Carcinoma of ovary	400 r	Good
9	63	Female	Carcinoma of rectum with metastases	600 r	Excellent
10	32	Female	Hodgkin's disease	600 r	Good
11	46	Female	Carcinoma of cervix	400 r	Good
12	45	Female	Carcinoma of rectum	650 r	Excellent
13	47	Female	Carcinoma of cecum	700 r	Good
14	28	Male	Marie-Strümpell arthritis	400 r	Excellent
15	48	Female	Carcinoma of cervix	600 r	Excellent
16	48	Male	Seminoma of testis	400 r	Excellent
17	45	Female	Carcinoma of bladder	300 r	Good
18	57	Male	Metastatic carcinoma of lungs	700 r	Excellent
19	57	Female	Carcinoma of uterus with metastasis	375 r	Excellent
20	40	Female	Carcinoma of bladder	400 r	Excellent
21	43	Female	Carcinoma of esophagus	300 r	Good
22	71	Female	Carcinoma of uterus	400 r	Excellent
23	45	Male	Carcinoma of prostate with metastases in spine and pelvis	675 r	Good
24	45	Female	Carcinoma of breast with generalized metastases	600 r	Poor
25	20	Male	Sarcoma metastatic to spine and lungs	600 r	Poor
26	40	Male	Marie-Strümpell arthritis	400 r	Poor
27	28	Female	Carcinoma of ovary	400 r	Good
28	56	Male	Carcinoma of rectum	400 r	Good
29	54	Female	Carcinoma of cervix	400 r	Excellent
30	40	Female	Carcinoma of cervix	400 r	Good
31	42	Female	Carcinoma of kidney	300 r	Excellent
32	33	Male	Marie-Strümpell arthritis	400 r	Poor
33	44	Female	Carcinoma of uterus	400 r	Good
34	23	Female	Endometriosis with abdominal metastasis	600 r	Good
35	40	Female	Carcinoma of cervix	400 r	Good
36	34	Female	Hodgkin's disease	350 r	Excellent
37	33	Male	Marie-Strümpell arthritis	400 r	Excellent
38	73	Male	Regional ileitis	150 r	Good
39	47	Female	Carcinoma of bladder	300 r	Excellent
40	44	Male	Seminoma of testis	400 r	Excellent
41	70	Female	Carcinoma of kidney	300 r	Excellent
42	48	Male	Hodgkin's disease	300 r	Excellent
43	14	Male	Grawitz tumor of kidney with metastasis	600 r	Good
44	77	Female	Carcinoma of ovary with abdominal metastasis	350 r	Good
45	73	Female	Carcinoma of breast	300 r	Excellent
46	58	Male	Carcinoma of rectum with metastases	400 r	Good
47	25	Male	Marie-Strümpell arthritis	400 r	Excellent
48	37	Male	Marie-Strümpell arthritis	400 r	Good
49	52	Female	Carcinoma of cervix with metastasis to pelvis	600 r	Excellent
50	27	Male	Marie-Strümpell arthritis	400 r	Good
51	12	Female	Virus pneumonia	150 r	Excellent
52	68	Female	Lymphosarcoma, retroperitoneal	200 r	Good
53	56	Male	Carcinoma of tonsil with metastasis to cervical nodes	200 r	Poor
54	63	Male	Rheumatoid arthritis	400 r	Poor
55	32	Male	Marie-Strümpell arthritis	400 r	Excellent
56	34	Female	Carcinoma of ovary	400 r	Excellent
57	75	Female	Carcinoma of gallbladder	300 r	Excellent
58	66	Female	Carcinoma of ovary	400 r	Excellent
59	71	Male	Inoperable carcinoma of rectum	400 r	Excellent

Case	Age	Sex	Diagnosis	Daily Dose of Radiation	Result of Treatment with Pyridoxine Hydrochloride
60	41	Female	Carcinoma of breast	300 r	Excellent
61	44	Female	Carcinoma of cervix and vagina	400 r	Excellent
62	52	Female	Carcinoma of ovary	400 r	Excellent
63	42	Female	Carcinoma of rectum	675 r	Excellent
64	39	Female	Carcinoma of cervix with extension to broad ligaments	400 r	Excellent
65	26	Male	Carcinoma of nasopharynx with metastasis to spine and pelvis	700 r	Good
66	45	Male	Carcinoma of rectum	400 r	Excellent
67	25	Male	Marie-Strümpell arthritis	400 r	Excellent
68	34	Female	Marie-Strümpell arthritis	450 r	Excellent
69	42	Female	Carcinoma of cervix with local metastasis	400 r	Good
70	33	Female	Carcinoma of cervix	400 r	Excellent
71	34	Female	Carcinoma of cervix with metastasis	650 r	Excellent
72	11	Female	Actinomycosis of lungs and pleura	150 r	Excellent
73	33	Female	Metastatic carcinoma of spine and abdomen	675 r	Good
74	61	Male	Carcinoma of bladder	400 r	Poor
75	64	Female	Carcinoma of breast with metastasis	650 r	Good
76	24	Female	Hodgkin's disease	600 r	Excellent
77	43	Female	Lymphoblastoma	600 r	Excellent
78	64	Female	Metastatic carcinoma of spine and pelvis	650 r	Poor
79	49	Female	Carcinoma of ovary, inoperable	650 r	Excellent
80	36	Female	Carcinoma of cervix	600 r	Excellent
81	50	Female	Carcinoma of kidney	300 r	Excellent

parts of the body possessing the largest capillary beds and, to combat it, they recommend administration of a vasoconstrictor such as benzedrine sulfate. The results obtained with this and a like drug were encouraging.

More recently, Ellinger (3) has called attention to the close correlation between the threshold dose of x-rays producing liver injury in the laboratory animal and sensitivity to histamine. He points out the close similarity of liver damage due to x-rays and that produced by injections of histamine, the histologic appearance of the liver cells being identical. An increase in bile secretion was also observed both after irradiation and histamine administration. Ellinger believes that he has proved experimentally that histamine is liberated in the tissues irradiated by x-ray and that it is to this histamine effect on the capillaries and liver that radiation sickness is attributable. If this theory is correct, it may well be possible to prove a direct relationship between histamine sensitivity in man and the unexplained occurrence of severe radiation sickness. We feel there is need for further experimentation in order to determine the mode of action of pyridoxine hydrochloride in the relief of radiation sickness.

CONCLUSIONS

Our experience in the use of pyridoxine hydrochloride in radiation sickness leads us to believe that it is a reliable and harmless method of relieving that condition.

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Pontiac General Hospital
Pontiac, Mich.

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Peroral Administration of Vitamin B_6 (Pyridoxine Hydrochloride) in the Treatment of Radiation Sickness[1]

A. OPPENHEIM, M.D., and BJORN LIH, M.D.[2]

A SERIOUS complication of radiation therapy is the frequent occurrence of nausea and vomiting, commonly called radiation sickness. At times this becomes so severe that it necessitates temporary or even permanent cessation of a projected course of therapy.

Since the individual reaction to radiation is variable, one cannot predict which patient will develop radiation sickness. Empirically, the determining factors should be the size of the port, dosage, and region treated, but such is not always the case. The amount of roentgen absorption necessary to produce and maintain radiation sickness has not been determined and is probably not a constant factor. It has been our experience, however, that ill effects seldom follow treatment of peripheral ports, while irradiation of the torso, particularly the abdomen, usually produces some nausea and vomiting. We have not noted any definite relationship between nutritional status and the ability to tolerate radiation therapy.

Many theories have been advanced as to the cause of radiation sickness, and many therapeutic measures have been tried, none of which has proved entirely satisfactory. The vitamin B group has been used extensively, and with not inconsiderable success. The use of vitamin B_6 by Willis *et al.* (1) in the nausea and vomiting of pregnancy and the uniformly good result obtained by Maxfield, McIlwain, and Robertson (2) from the intravenous administration of this drug in the treatment of radiation sickness prompted our present investigation. The intravenous route of administration, although effective, is cumbersome, time-consuming, and less suitable to a large clinic than the peroral route. This paper is a preliminary report on the use of vitamin B_6 (pyridoxine hydrochloride) given *per os* to a series of 50 patients who developed nausea or vomiting, or both, during radiation therapy, or had had these symptoms during a previous course of treatment. Otherwise the patients were unselected.

After preliminary investigation, it was found that the best results were obtained when 75 to 100 mg. doses were given one-half hour before meals, three or four times a day. The larger doses were chosen when treatment was given to the abdomen or chest, the smaller when treatment was directed to the pelvis or peripheral ports, such as the axilla, neck, or groin. If the dose was less than 75 mg., the maximal benefit was not obtained. Occasionally, when the drug was not retained because of vomiting, the oral dose was supplemented by intravenous medication.

Table I shows the results of peroral administration of vitamin B_6 in the 50 cases. Unless otherwise specified, the irradiation factors were: 250 kv., 1.5 mm. copper filter, 50–70 cm. target-skin distance. The results were graded as follows: *Excellent* (*E*) when there was complete cessation of nausea and vomiting; *Good* (*G*) when the patient had only occasional days of distress, the symptoms being reduced at least 75 per cent by medication; *Fair*, when symptoms were reduced only 50 per cent; and finally, *Failure*, when symptoms were slightly or not at all relieved. Before the pills were given, the patients were told that the medication was for experimental purposes, and each was asked to keep a daily record of the degree of nausea, vomiting, or any other untoward symptoms that oc-

[1] Accepted for publication in February 1946.

[2] Both authors trainees of the National Cancer Institute in diagnosis and treatment of cancer, at the Memorial Hospital, New York.

TABLE I: RESULTS OF VITAMIN B_6 THERAPY IN 50 CASES OF RADIATION SICKNESS

Patient Age Sex	Diagnosis	Factors of X-Ray Therapy: Ports, with Size in cm.	Daily Dose, r	Total Dose, r	Dose of B_6	Result	Remarks
C. D. M 62	Carcinoma of nasopharynx	Peroral....3.0	200–300	4,900	75 mg. t.i.d.	E	
		L. nasal....2.5	500	8,000			
		Supra-orbital....2.5	500	4,500			
		R. zygoma....6 × 4	250	2,000			
Y. H. H. F 32	Carcinoma of nasopharynx	R. and L. maxilla....5 (circle)	250	4,500 × 2	75 mg. t.i.d.	E	
		L. upper neck....5 (circle)	250	4,500			
		R. zygoma....5 (circle)	250	4,500			
E. H. F 76	Carcinoma of nasopharynx	Peroral....3.5	300	4,200	75 mg. t.i.d.	E	
		L. cheek....5.0	300	3,000			
		R. cheek....5.0	300	3,000			
		L. zygoma....5.0	300	900			
		R. zygoma....5.0	300	600			
E. B. F 51	Carcinoma of breast (postop.)	Axilla			75 mg. t.i.d.	E	
		Ant....12 × 14	250	2,000			
		Post....19 × 12	250	2,000			
		Dir....13 × 21	250	1,750			
		(Two treatments daily)					
		Chest					
		Lat....17 × 11	500	1,500			
		Med....27 × 12	500	1,500			
		(Low voltage)					
M. P. F 48	Carcinoma of breast (postop.)	As in preceding case			75 mg. t.i.d.	E	Placebo given for 1 week without benefit
M. W. F 43	Carcinoma of breast (postop.)	As in preceding case			75 mg. q.i.d.	E	
E. P. F 47	Carcinoma of breast (recurrent)	Axilla, dir....23 × 14	300	1,800	75 mg. t.i.d.	Failure	Symptoms aggravated
		Chest, ant. (low voltage)....23 × 13	300	1,800			
		(One treatment daily)					
A. H. F. 26	Hodgkin's disease	Mediastinum			75 mg. t.i.d.	E	
		Ant....17 × 12	300	1,800			
		Post....18 × 12	300	1,800			
		(One port treated daily)					
C. A. F 37	Hodgkin's disease	Mediastinum			75 mg. t.i.d.	E	
		Ant....15 × 11	100	400			
		Post....15 × 11	100	400			
		Chest, lat....12 × 9	200	800			
		(One port treated daily)					
M. A. W. F 20	Hodgkin's disease	Mediastinum			75 mg. t.i.d.	E	
		Ant....14 × 8	200	800			
		Post....19 × 6	200	800			
		Chest, lat....11 × 10	200	800			
M. B. M 25	Hodgkin's disease	L. neck....15 × 11	200	1,800	75 mg. t.i.d.	G	
		R. neck....15 × 14	200	1,800			
		Mediastinum					
		Ant....13 × 8	200	1,800			
		Post....13 × 9	200	1,800			
		(One neck and one mediastinal port treated daily)					
E. S. F 53	Lymphosarcoma	R. axilla....10 × 8	300	900	75 mg. t.i.d.	G	
		L. axilla....12 × 11	300	900			

		Mediastinum					
		Ant.	13 × 8	200	1,800		
		Post.	13 × 9	200	1,800		
		(One neck and one mediastinal port treated daily)					
E. S. F 53	Lymphosarcoma	R. axilla	10 × 8	300	900	75 mg. t.i.d.	G	
		L. axilla	12 × 11	300	900			
		R. lower chest						
		Ant.	19 × 18	400	800			
		Post.	13 × 17	400	800			
		R. upper chest						
		Ant.	11 × 11	400	800			
		Post.	13 × 19	400	800			
		R. chest, lat.	16 × 12	400	800			
		L. neck	5 (circle)	300	1,800			
B. W. M 53	Carcinoma of esophagus with gastrostomy	Epigastrium				100 mg. q.i.d.	Fair	
		Ant.	16 × 16	300	2,400			
		Post.	18 × 16	300	2,400			
M. G. M 63	Carcinoma of esophagus	Two ant. ports	18 × 8	400	2,400 × 2	75 mg. q.i.d.	E	
		Two post. ports	25 × 8	400	2,400 × 2			
F. S. M 66	Carcinoma of lung	R. upper chest				75 mg. t.i.d.	G	Constant vomiting during a previous similar series. B_6 started at beginning of present series
		Ant. and post. (1,000 kv.)	15 × 10 (oval)	300	900 × 2			
S. A. M 45	Carcinoma of lung	As in preceding case		300	4,200 × 2	100 mg. t.i.d.	E	
C. H. M 51	Carcinoma of lung	Right apex				75 mg. t.i.d.	E	Last 3 treatments on 250-kv. machine
		Ant. and post. (1,000 kv.)	10 (circle)	...	3,600 × 2			
J. M. M 46	Carcinoma of lung	R. supraclavicular	9 (circle)	350	2,450	75 mg. t.i.d.	E	
		R. chest: Ant. and post.	10 × 9	300	2,100 × 2			
H. B. M 59	Carcinoma of lung	L. chest: Ant. and post. 1,000 kv.	10 × 15	300	3,000 × 2	75 mg. t.i.d.	E	
V. A. F 11	Sarcoma of lung	Two ant. chest ports	10 × 10	200	2,400 × 2	75 mg. t.i.d.	G	
		Two post. ports	12 × 12	200	2,400 × 2			
		(Two ports treated daily)						
J. M. M 31	Carcinoma of testis, metastatic to lung	L. chest				75 mg. t.i.d.	E	
		Ant., post., lat.	19 × 11	500	2,000 × 3			
J. D. M 40	Lung metastasis, primary unknown	R. chest				75 mg. t.i.d.	E	100 mg. intravenously before first treatment
		Ant. and post.	17 × 14	200	600 × 2			
A. T. F 57	Carcinoma of breast with metastases	Sternum	12 × 16	250	3,000	75 mg. q.i.d.	E	No medication last 10 days; constant vomiting
		R. axilla	14 × 13	300	1,500			
		R. pelvis	30 × 18	400	1,200			
L. M. F 58	Carcinoma of stomach	Stomach: Ant. and post.	19 × 17	300	1,800 × 2	75 mg. q.i.d.	Fair	
L. C. M 63	Carcinoma of stomach	Stomach: Ant. and post.	13 × 13	200–250	1,900 × 2	100 mg. q.i.d.	Fair	
A. K. M 50	Carcinoma of stomach	Stomach: Ant. and post.	20 × 15	300	2,400 × 2	100 mg. q.i.d.	Failure	
H. S. M 77	Carcinoma of stomach	Stomach: Ant. and post.	18 × 21	300	2,700 × 2	100 mg. q.i.d.	Fair	
P. C. M 22	Hodgkin's disease	Mid-abdomen	17 × 18	400	1,600	75 mg. q.i.d.	Fair	Symptoms only when upper abdomen was treated
		R. lower abdomen	18 × 16	400	1,600			
S. W. M	Hodgkin's disease	Central abdominal ports				75 mg. t.i.d.	Fair	
		2 ant., 2 post.	13 × 12	200	800 × 4			
J. H. M 46	Hodgkin's disease	L. neck	19 × 12	300	900	75 mg. t.i.d.	E	
		Central trunk						
		3 ant., 3 post.	17 × 9	200	4,400 × 6			
M.S. F 23	Hodgkin's disease with bone involvement	R. ilium				75 mg. t.i.d.	E	
		Ant.	13 × 11	300	900			
		Post.	19 × 17	300	900			(Record of case cont. on p. 384)

TABLE I: RESULTS OF VITAMIN B_6 THERAPY IN 50 CASES OF RADIATION SICKNESS (*Continued*)

Patient Age Sex	Diagnosis	Factors of X-Ray Therapy: Ports, with Size in cm.	Daily Dose, r	Total Dose, r	Dose of B_6	Result	Remarks
M. S. (*Cont.*)		Central abdomen Ant. and post. 12 × 12 (One ilial and one abdominal port treated daily)	300	900 × 2			
J. R. F 47	Lymphosarcoma	Epigastrium Ant. and post. 19 × 9	100	200 × 2	75 mg. t.i.d.	E	
		R. and L. axilla. 12 × 10	300	600 × 2			
V. T. M 31	Lymphosarcoma	R. and L. lower quadrants. 17 × 12	100–300	1,450 × 2	75 mg. t.i.d.	E	
		Central abdomen Ant. and post. 20 × 8	200	600 × 2			
		Spleen: Ant. and post. 12 × 9	100	300 × 2			
		R. groin. 12 × 16	300	900			
M. B. F 28	Lymphosarcoma	8 abdominal ports. 13 × 7 to 19 × 11	75–200	5,800	100 mg. q.i.d.	Failure	
R. F. F 42	Carcinoma of sigmoid, inoperable; colostomy	R. and L. lower quadrant. 15 × 15	300	2,100 × 2	75 mg. q.i.d.	Fair	
L. O. F 39	Carcinoma of breast metastasizing to bone	Dorsal spine. 18 × 13	350	1,400	100 mg. t.i.d.	G	
		Lumbar spine. 16 × 13	350	1,400			
		4 pelvic ports. 20 × 20	400	1,200 × 4			
H. B. M 31	Teratoma testis	L. groin and scrotum. 17 × 12	500	1,000	100 mg. q.i.d.	Failure	
		Abdominal ports 2 ant. and 2 post. 18 × 12 (Two abdominal ports treated daily)	300	2,100			
G. W. F 38	Carcinoma of ovary	4 pelvic and low abdominal ports. 17 × 11 (Two ports treated daily)	250	2,000 × 4	75 mg. q.i.d.	E	Daily vomiting during previous series
G. B. F 53	Carcinoma of ovary	4 pelvic and 4 abdominal ports (1,000 kv.). 16 × 12	250 × 2	1,000 × 8	75 mg. t.i.d.	G	
F. L. F 43	Carcinoma of ovary	4 pelvic and 4 abdominal ports (1,000 kv.). 14 × 12	250 × 2	1,000 × 8	100 mg. t.i.d.	E	
T. S. F 40	Carcinoma of ovary	R. ant. abdomen. 23 × 16	300	1,800	75 mg. q.i.d.	E	
		L. ant. abdomen. 19 × 19	300	1,800			
B. Z. F 34	Carcinoma of ovary	R. and L. lower abdomen. 23 × 15	300	2,100 × 2	75 mg. t.i.d.	Failure	
M. W. F 39	Carcinoma of fundus (postop.)	6 pelvic ports. 14 × 11	250 × 2	1,000 × 6	75 mg. t.i.d.	Failure	Rash developed
F. E. F 63	Carcinoma of fundus	4 pelvic ports. 17 × 13	250 × 2	2,000 × 4	100 mg. t.i.d.	E	
S. M. F 38	Carcinoma of cervix	4 pelvic ports. 16 × 12	250 × 2	1,750 × 4	75 mg. t.i.d.	Fair	
B. M. F 45	Carcinoma of cervix	4 pelvic ports. 15 × 12	250 × 2	2,000 × 4	75 mg. t.i.d.	E	
I. J. F 34	Carcinoma of cervix	6 pelvic ports. 14 × 11	250 × 2	2,000 × 6	75 mg. t.i.d.	E	
G. M. F 41	Carcinoma of cervix	6 pelvic ports. 14 × 11	250 × 2	1,750 × 6	75 mg. t.i.d.	E	
E. B. F 43	Carcinoma of cervix	6 pelvic ports. 14 × 11	250 × 2	1,750 × 6	75 mg. t.i.d.	E	
R. P. F 46	Carcinoma of vagina	6 pelvic ports. 17 × 12	250 × 2	2,250 × 6	75 mg. t.i.d.	E	

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curred. They were seen frequently during the period of observation. An effort was made to evaluate the pharmacological action of the drug as distinct from the psychological effect manifest in taking pills, keeping of records, and the personal attention involved.

From Table I, it will be seen that in 30 cases (60 per cent) results were excellent; in 6 (12 per cent) good; in 8 (16 per cent) fair, while there were 6 (12 per cent) failures. Five of the 6 failures, as shown in Table II, occurred when the abdomen was irradiated, although 8 of the 21 cases so treated showed excellent results. This confirms our previous observation that treatment over the abdomen is more apt to produce nausea and vomiting than irradiation of any other body region, and the symptoms are more difficult to control with vitamin B_6.

It was found that those who failed to respond to the initial dosages calculated for the various body regions did not improve even though larger doses were given. On the other hand, a number of patients, once nausea and vomiting were controlled, were able to continue treatment for some time without pills, and then required lesser amounts for control of recurrent symptoms.

In a few cases, a placebo was substituted for the vitamin B_6 after symptoms had been alleviated by the latter drug. Symptoms always recurred, but were controlled again with resumption of the vitamin administration.

No significant alteration in blood pressure was noted.

The symptoms of radiation sickness were aggravated in one patient following the administration of vitamin B_6. In one patient a scaly and follicular type of eruption developed, which disappeared on withdrawal and reappeared on readministration of the drug.

TABLE II: RESULTS OF VITAMIN B_6 THERAPY IN 50 CASES OF RADIATION SICKNESS SUMMARIZED

Body Region Treated	Number of Cases	Number Excellent	Number Good	Number Fair	Number, Failures
Head, Neck	3	3	0	0	0
Breast	4	3	0	0	1
Chest	16	11	4	1	0
Abdomen	21	8	2	6	5
Pelvis	6	5	0	1	0
TOTAL	50	30 (60%)	6 (12%)	8 (16%)	6 (12%)

CONCLUSION

A preliminary report is presented on the peroral use of vitamin B_6 (pyridoxine hydrochloride) in the treatment of radiation sickness in a series of 50 cases. A significantly high percentage of favorable results was obtained.

ACKNOWLEDGMENTS: The authors are indebted to Merck & Co., Inc., Rahway, N. J., for the vitamin B_6 (pyridoxine hydrochloride) used in this study. They are also grateful to the attending physicians of the various services at Memorial Hospital for furnishing them with the clinical material, and to the technicians of the Radiation Department for their co-operation.

Memorial Hospital
444 East 68th St.
New York 21, N. Y.

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Pyridoxine Hydrochloride (Vitamin B_6) in the Control of Radiation Sickness: Preliminary Report[1]

LAWRENCE D. SCOTT, M.D.,[2] and GADSON J. TARLETON, M.D.[3]

Nashville, Tenn.

SINCE 1934, we have used the protracted fractionated method of deep x-ray therapy in the treatment of practically all types of malignant neoplasms that have been referred to us for treatment. As in most general radiological clinics, carcinomas of the uterine cervix and breast are by far the most frequent types of lesion encountered. From 1934 to 1941, we used the old Drum type of equipment unit, operating at 200 kv.p., 5 ma., 50 cm. distance, 0.5 mm. Cu plus 1.0 mm. Al filter, with an output of 17 r per minute in air, not including back-scatter. During that seven-year period, the control of x-ray sickness was quite a problem. We used face masks (1), nembutal (2), fruit juices (3), good ventilation of therapy rooms (4), and many other measures recommended by various authors, with variable results.

In 1941, we installed a modern shock-proof 220-kv.p. unit, operating at 200 kv., 20 ma., 50 cm. distance, and an r output of 50 per minute, with 0.5 mm. Cu plus 1.0 mm. Al filtration. From that time on, radiation sickness ceased to be a major problem unless we increased our daily dose from 300 to 500 or 600 r. It was about this time that we began to administer nicotinic acid (5) and thiamine chloride (6) routinely to patients receiving 200 kv. x-ray therapy. This probably helped to prevent some cases of radiation sickness, but in other instances it was wholly ineffective.

It is generally recognized that the larger the daily dose of radiation with a 200-kv. unit, the greater is the possibility of x-ray sickness. The size of the field, the area treated, and the general nutritional state of the patient also play important roles (7). It has been our experience that when two areas of the pelvis or breast are treated, with fields up to 15 × 15 cm. and a daily dosage of 150 r measured in air to each area, severe radiation sickness rarely develops, provided proper attention is given to the patient's general nutritional state (8). Diarrhea practically never occurs under these circumstances. If, however, the epigastric area or right and left hypochondriac regions are treated with 150 r, using 15 × 15 cm. fields, radiation sickness occurs much more frequently and usually requires special attention if therapy is to be continued (see Case 18). Lesions of the extremities can be given large doses of radiation without signs of radiation sickness (see Case 5).

In April 1945, we decided to double the daily x-ray dosage of 300 r, so as to give 600 r. In this way we hoped to decrease the time necessary for adequate treatment of cases of carcinoma in the hospital, to make possible a more rapid turnover of cases, and to reduce the hospital expenses proportionately.

Economics plays an important part in the management of any type of malignant disease, especially among people whose average economic status is low. Two questions immediately confronted us: (1) How could we control the x-ray sickness that we felt would most probably develop? (2) Would the end-results be equally good? It is the purpose of this paper to answer the first question. Obviously, we cannot answer the second until more time has elapsed

TABLE I: PROPHYLACTIC USE OF PYRIDOXINE HYDROCHLORIDE (VITAMIN B_6) FOR RADIATION SICKNESS: REVIEW OF 20 CASES

Name, Age Sex, Date	Diagnosis	X-Ray Treatments	Vit. B_6 Intravenously and 50 mg. Nicotinic	Symptoms: Nausea	Vomiting	Diarrhea	Appe-	Others	Results

[1] From the Department of Radiology, George W. Hubbard Hospital of Meharry Medical College, Nashville, Tenn. Accepted for publication in April 1946.

[2] Chairman of the Department of Radiology and Professor of Radiology of Meharry Medical College; Radiologist, George W. Hubbard Hospital.

[3] Teaching Fellow in Radiology, Meharry Medical College.

TABLE I: PROPHYLACTIC USE OF PYRIDOXINE HYDROCHLORIDE (VITAMIN B_6) FOR RADIATION SICKNESS: REVIEW OF 20 CASES

Name, Age Sex, Date X-Ray Therapy Begun	Diagnosis	X-Ray Treatments	Vit. B_6 Intravenously and 50 mg. Nicotinic acid t.i.d.	Symptoms					Results
				Nausea	Vomiting	Diarrhea	Appetite	Others	
1. L. W. 69 F 7/30/45	Carcinoma of right breast	Supra and inframammary regions and posterior part of right axilla. Areas 15 × 15 cm.; 150 r to 2 areas for 2 days. Later, 300 r to each area, 5 days weekly. Total: 1,800 r to each area	8/6/45 25 mg. B_6	8/6/45	No	No	Fair	None	Complete relief 8/6. No symptoms after that date
2. E. M. W. 41 F 11/5/45	Carcinoma of cervix	Pelvis: anterior and posterior regions, right and left. Areas 15 × 15 cm.; 300 r to 2 areas, 5 days weekly. Total: 1,650 r to ant. and 1500 r to post. regions	25 mg. B_6 immediately after each treatment	No	No	No	Normal	None	No nausea, vomiting, or other symptoms
3. V. C. 32 F 8/1/45	Carcinoma of cervix	Pelvis: anterior and posterior regions, right and left. 300 r to 2 areas, 15 × 15 cm., 5 days weekly. Total: 1,500 r to each area	8/1/45 25 mg. B_6	No	No	No	Fair	None	No nausea, vomiting, or other symptoms
4. W. F. 40 F 8/8/45	Carcinoma of cervix	Pelvis: anterior and posterior regions right and left. Areas 15 × 15 cm.; 300 r to 2 areas, 5 days weekly. Totals: 1,800 r to anterior regions; 1,500 r to posterior regions	8/21/45 25 mg. B_6	Slight 8/21	No	No	Fair	None	Nausea relieved completely 8/21 after administration of vitamin. No symptoms after that
5. S. S. 51 F 8/21/45	Sarcoma of left leg	Left leg and thigh: 300 r to 2 areas, 15 × 15 cm., 5 days weekly. Total: 2,100 r to leg; 2,400 r to thigh	25 mg. B_6 immediately after each treatment	No	No	No	Fair	Pain in left extremity	No nausea, vomiting, or other symptoms
6. D. W. R. 59 M 9/12/45	Carcinoma of prostate with metastasis to urinary bladder	Hypogastric region, right and left side, anterior and posterior. Areas 10 × 10 cm. anterior; 15 × 15 cm. posterior; 300 r to 2 areas. Total: 2100 r anterior regions; 1,500 r posterior regions	25 mg. B_6 immediately after each treatment	No	No	Slight 9/26	Good	None	Diarrhea controlled with bismuth subcarbonate

Table cont. on p. 388

TABLE I: PROPHYLACTIC USE OF PYRIDOXINE HYDROCHLORIDE (VITAMIN B_6) FOR RADIATION SICKNESS: REVIEW OF 20 CASES (*Continued*)

Name, Age, Sex, Date X-Ray Therapy Begun	Diagnosis	X-Ray Treatments	Vit. B_6 Intravenously and 50 mg. Nicotinic Acid t.i.d.	Symptoms					Results
				Nausea	Vomiting	Diarrhea	Appetite	Others	
7. E. M. 39 F 10/1/45	Carcinoma of cervix	Pelvis: left and right, anterior and posterior; 300 r to 2 areas, 15 × 15 cm., 5 days weekly. Total: 1,500 r to each area	25 mg. B_6	No	No	No	Good	None	No nausea, vomiting, or other symptoms
8. L. L. 52 F 10/9/45	Carcinoma of cervix	Pelvis: anterior and posterior regions, right and left. Areas 15 × 15 cm.; 300 r to 2 areas, 5 days weekly. Total: 1,500 r anterior regions; 1,200 r posterior regions	25 mg. B_6 immediately after each treatment	No	No	No	Good	None	No nausea, vomiting, or other symptoms
9. S. W. 50 F 10/13/45	Carcinoma of cervix	Pelvis: anterior and posterior regions, left and right. Areas 15 × 15 cm.; 300 r to 2 areas, 5 days weekly. Total: 1,500 r to each area	25 mg. B_6 immediately after each treatment	No	No	No	Good	None	No nausea, vomiting, or other symptoms
10. A. W. 58 M 10/16/45	Carcinoma of rectum	Pelvis: anterior and posterior regions, right and left; 200 r to 2 areas, 10 × 15 cm., posterior region, alternating with 300 r to one anterior area, 5 days weekly. Total: 1,500 r to each region	25 mg. B_6 immediately after each treatment	No	No	No	Good	None	No nausea, vomiting, or other symptoms
11. H. P. 31 M 10/17/45	Sarcoma of buttocks	300 r to one area, 15 × 15 cm., on each buttock 17th and 18th days. Later, 300 r to left buttock, 5 days weekly; 600 r to right buttock 23rd day. Total: 1,200 r to right and 1,800 r to left buttock	25 mg. B_6 immediately after each treatment	No	No	No	Good	Pain at site of tumor	No nausea, vomiting, or other symptoms, except pain at site of tumor
12. A. J.	Carcinoma of	Inframammary region	25 mg. B_6 im-	No	No	No	Fair	None	No nausea vomiting, or other

12.	A. J. 76 F 10/17/45	Carcinoma of left breast with metastasis to axilla	Inframammary region and left axilla treated alternately with 300 r to each area, 10 × 10 cm., up to 10/24. Later, both regions treated 5 days weekly. Total: 1,500 r to each region. One area, 7 × 7 cm., in the left supraclavicular region up to 10/-24. Total: 1,800 r	25 mg. B_6 immediately after each treatment	No	No	No	Fair	None	No nausea vomiting, or other symptoms
13.	M. L. W. 33 F 10/21/45	Carcinoma of cervix	Pelvis: right and left, anterior and posterior; 300 r to 2 areas, 15 × 15 cm., 5 days weekly up to 25th day. Later, 150 r to 2 areas, 5 days weekly up to 11/3. Total: 1,800 r to anterior regions; 1,200 r to posterior regions	25 mg. B_6 immediately after each treatment	No	No	No	Fair	General weakness and indisposition	The large doses were discontinued 10/-27, because the patient began to have a fever of 102 to 106° F. during the days preceding the treatment. The fever ceased when the treatment was suspended on the 27th and 28th days, and did not recur after treatments were resumed with 150 r to both areas, 5 days weekly
14.	T. M. 45 M 10/27/45	Carcinoma of stomach	Epigastrium: right and left regions. Areas 10 × 15 cm. treated on 27th day; areas 10× 10 cm. treated 28th day; 300 r to 2 areas for 2 days	25 mg. B_6 immediately after each treatment	Moderate but persistent	No	No	Good	Indisposition and weakness	The nausea was not completely relieved, but patient, who also complained of gas in the stomach, improved after taking B_6
15.	E. A. D. 18 M 11/1/45	Papilloma of urinary bladder	Vesical region: anterior, right and left; 300 r to 1 area, 10 × 10 cm., 5 days weekly. Total: 1,500 r to each area	25 mg. B_6 immediately following treatments on 11/-8, 11/10, 11/-12, 11/13	Moderate 11/8	No	No	Fair	None	Nausea permanently relieved
16.	A. A. 39 F 11/5/45	Carcinoma of cervix	Pelvis: anterior and posterior regions, right and left. Areas 10 × 15 cm., 150 r to 2 areas, 5 days weekly. Total: 900 r to anterior region; 750 r posterior	25 mg. B_6 immediately after the treatments on 11/-5 and 11/6; 50 mg. from 7th day on	Slight 11/5 and 11/16. Moderate 11/7. Absent thereafter	Moderate 11/5. Severe 11/6. Absent thereafter	No	Fair	None	Nausea permanently relieved after 11/7

Table cont. on p. 390

TABLE 1: PROPHYLACTIC USE OF PYRIDOXINE HYDROCHLORIDE (VITAMIN B_6) FOR RADIATION SICKNESS: REVIEW OF 20 CASES (*Continued*)

Names, Age, Sex, Date X-Ray Therapy Begun	Diagnosis	X-Ray Treatments	Vit. B_6 Intravenously and 50 mg. Nicotinic Acid t.i.d.	Symptoms					Results
				Nausea	Vomiting	Diarrhea	Appetite	Others	
17. A. C. N. 44 F 11/12/45	Carcinoma of right breast with metastasis to right axillary nodes	Superior and inferior regions of breast and posterior axillary region; 300 r to 2 areas, 15 × 15 cm., 5 days weekly. Total: 1,500 r to each area	25 mg. B_6 immediately after treatment 11/13; 50 mg. 11/14 and after	Moderate 11/13; slight, 11/14	No	No	Fair	None	Partial relief of nausea 11/13; permanent and complete relief 11/14
18. F. W. 70 F 11/23/45	Carcinoma of left breast with metastasis to liver	Superior and inferior left breast and posterior left axilla; 300 r to 2 areas, 15 × 15 cm., 5 days weekly. Total: 900 r to each region. Superior, inferior, right and left, on upper part of abdomen. Total: 900 r anterior and 750 posterior, to each area, respectively	25 mg. B_6 immediately after treatment on 12/19; 50 mg. 12/20; 75 mg. 12/21; 125 mg. 12/22; 50 mg. 12/26 and 12/27	Severe	Absent until 12/23; then daily until 12/28. Absent thereafter	Constipation; then normal stools; then diarrhea	Poor	Pain in epigastrium; constipation except one day	Nausea partially relieved immediately with B_6. Vomiting occurred as soon as B_6 was suspended, 12/23. Diarrhea relieved with subcarbonate of bismuth
19. P. L. 38 F 12/6/45	Round-cell sarcoma of abdomen	Superior, inferior, right and left anterior abdomen; 300 r to 2 areas, 15 × 15 cm., 5 days weekly. Total: 1,800 r to each area	25 mg. B_6 immediately after each treatment except 12/7	Slight 12/10	No	No	Good	Constipation with tendency to progressive increase in frequency of stools	Slight nausea 12/10
20. M. J. M. 41 F 12/12/45	Carcinoma of cervix	Anterior and posterior, right and left pelvis; 300 r to 2 areas 15 × 15 cm., 5 days weekly. Total: 1,550 r to anterior regions; 1,350 r to posterior regions	25 mg. B_6 12/14 only	Moderate 12/14	No	No	Good	None	Complete and permanent relief of nausea 30 minutes after administration of B_6, 12/14. No other symptoms on or after that day

and comparisons can be made with other groups treated with smaller dosages. Kaplan (9), however, recommends 1,000 to 2,000 r in from ten days to two weeks as pre-radium x-ray therapy in carcinoma of the cervix. This became the basis of our modified massive dose technic. The four breast cases included in our chart were all inoperable and hopeless, and radiation therapy was given only for palliation.

Maxfield, McIlwain, and Robertson (10) reported excellent results with pyridoxine hydrochloride (vitamin B_6) in over 50 cases of radiation sickness. We decided to use this drug in addition to 50 mg. of nicotinic acid t.i.d. We also decided to give 25 mg. of pyridoxine hydrochloride intravenously immediately after each x-ray treatment, without waiting for symptoms to develop as the above authors had done.

Up to the present, we have used an x-ray dose of 600 r together with 25 mg. of pyridoxine hydrochloride, administered intravenously, in about 35 cases. The accompanying table shows the results in the first 20 cases (in one of these only 300 r per treatment were given). The majority of these patients were treated five times weekly, allowing them to rest on Saturday and Sunday. Up to the present the clinical response has been the same as when only 300 r were given, except that more cases of diarrhea have occurred. Treatment with a few doses of bismuth subcarbonate was effective in controlling that symptom in practically all instances.

All patients included in this report were American Negroes. Whether race is a factor in the frequency of radiation sickness is beyond the scope of this paper, but we have never had the high incidence of radiation sickness (50 per cent) reported by some writers (11).

In 5 of the 20 reported cases the patients were ambulatory and we felt that more than ordinary care had to be exercised in this group. Each of these patients was carefully questioned every day regarding nausea, vomiting, anorexia, diarrhea, or other suggestive symptoms. Only one reported some nausea and vomiting (Case 16). Incidentally, this is the patient who received 300 r per treatment, mentioned above. With increase of the dosage of pyridoxine hydrochloride to 50 mg. she experienced no further trouble.

SUMMARY

By increasing the daily x-ray dosage from 300 to 600 r and administering pyridoxine hydrochloride, we succeeded in reducing the treatment time for various types of malignant growth by approximately one-half and at the same time prevented the development of radiation sickness.

NOTE: The pyridoxine hydrochloride used in this investigation was kindly furnished by E. R. Squibb & Sons.

Meharry Medical College
Nashville 8, Tenn.

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Treatment of Radiation Sickness with Trasentine and Trasentine-Phenobarbital: Preliminary Report[1]

JOHN C. GLENN, JR., M.D., and ROBERT J. REEVES, M.D.
Durham, N. C.

IT IS WELL KNOWN that x-ray therapy injures healthy as well as malignant cells. According to Kallet and Thorstad (10), this injury is anatomically manifested by an initial edema as a result of damage to connective tissue and blood vessels. With further trauma, an endarteritic process develops, with thickening of the walls and a gradual decrease in the size of the lumen of the intestine.

The most distressing symptoms which follow intensive irradiation are nausea (sometimes vomiting) and diarrhea, frequently accompanied by pain and evidence of urinary bladder irritation. Such symptoms are seen particularly in those patients receiving radiation to the pelvic area, because of the anatomic proximity of the lower intestine to the uterus and the frequency with which x-ray therapy is employed for the treatment of carcinoma in or about the latter organ.

The cause of these sequelae remains an enigma, although it seems possible that they may be due to a direct action of the x-rays on the rectum and/or sigmoid colon, urinary bladder, and probably some portions of the small bowel.

Many factors, ranging from the psychic effect of the mechanical apparatus employed, with its attendant noise and accompanying odors, to blood electrolyte alterations and vitamin deficiencies, have been offered as explanations. Goldman (5) has described disturbances in both nitrogen and inorganic chloride balance and has shown that changes in the latter are associated with irradiation sickness, while Bean, Spies, and Vilter (1) reached the conclusion that persons on an inadequate diet are more prone to have untoward symptoms following x-ray therapy than are those who are adequately nourished. Mitchell (14) has described nucleic acid and carbohydrate metabolism alterations, and, though he does not correlate these with radiation sickness, it is certainly within the realm of possibility that they may be of importance in that respect. On this basis, several reports, (2, 4, 7, 11, 12, 13, 16) have appeared in the literature mentioning the use of vitamin supplements, particularly thiamine chloride, pyridoxine, and nicotinic acid. Jenkinson and Brown (8) have commented on the possible value of amphetamine and *d*-desoxyphedrine. On the other hand, Holmes (6) has demonstrated that serum cholesterol levels roughly parallel the development of radiation sickness, and that those patients in whom symptoms develop during the course of irradiation therapy show a fall in the cholesterol level.

Wallace (17) reported ileal studies on a series of patients receiving x-ray therapy over the pelvis and described changes occurring in the small bowel which simulated those of vitamin deficiency. It is interesting to note that he called attention to segmentation of the barium stream, diminution of intestinal motility, and narrowing of the lumen, as well as flattening of the mucosal pattern of the bowel. These observations, coupled with the well known abdominal cramps and tenesmus, strongly suggest that the immediate cause of the nausea, diarrhea, and abdominal pain may be intestinal and sphincter spasm. With this possibility in mind, we theorized that adequate antispasmodic therapy should provide at least symptomatic relief.

CASES AND TREATMENT

Sixty-five patients receiving intensive x-ray therapy are included in this preliminary series. There was no attempt at

[1] From the Department of Radiology, Duke University School of Medicine and Duke Hospital, Durham, N. C.
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selection of patients, except that those whose symptoms could be "cured" by smelling aromatic spirits of ammonia or other such medication were excluded.

Patients with carcinoma of the cervix were treated with 200-kv. x-rays through six external ports, with the addition of intravaginal therapy in a few instances. Those with carcinoma of the colon (all of them rectal lesions) received 400-kv. therapy through one anterior and two posterior ports over the involved area, except in case 31, in which radiation was given over the liver for metastasis. For Marie-Strümpell arthritis, 140-kv. therapy was used, directed to three spinal and sacroiliac ports. Carcinoma of the breast was treated through one anterior and one posterior port in the axillary regions, with 200-kv. therapy. Patients with Hodgkin's disease, lymphosarcoma, undifferentiated carcinoma, etc., received appropriate therapy over the indicated areas with 140- or 200-kv. x-rays.

The antispasmodic drug, trasentine (75 mg. per tablet), was used initially on 26 patients, with a dosage range from 225 to 1,000 mg. per day in divided doses. Thirty-two patients were then treated with trasentine-phenobarbital in doses of 1 or 2 tablets three times a day. Inasmuch as a trasentine-phenobarbital tablet contains 20 mg. of trasentine plus 20 mg. of phenobarbital, and since a trasentine tablet alone contains 75 mg. of the drug, it was thought that nausea and diarrhea might be controlled even more effectively if additional antispasmodic therapy were provided along with the phenobarbital. A small group of patients, therefore, complaining principally of those symptoms were treated with trasentine-phenobarbital, 1 tablet three times a day, plus trasentine in doses of 1 to 3 tablets (75 mg. each) three or four times a day. Results in this small series of 7 patients were gratifying.

This form of medication was selected because tablets are easy for most people to take, and the necessity of parenteral therapy is eliminated. Trasentine is non-habit-forming, and with the amount of phenobarbital contained in trasentine-phenobarbital tablets, the danger of barbiturate habituation is reduced to a minimum. Trasentine is an effective intestinal antispasmodic as shown by a number of workers (18). It has all of the desirable actions of both atropine and papaverine but is essentially devoid of the undesirable side-effects of the former drug on the heart, pupil, accommodation, and the salivary glands (3, 9, 15).

DISCUSSION

As shown in the accompanying table, 6 patients failed to gain relief from the use of either trasentine or trasentine-phenobarbital. Two of these had carcinoma of the cervix in terminal stages. One had recently been operated upon for carcinoma of the rectum and an ileostomy had been performed. In 3 patients who failed to respond to trasentine, diarrhea was also unrelieved by large doses of tincture of opium.

Five patients either stopped taking the drug after symptoms were relieved or consumed their entire supply and went for a time without obtaining more. Each of these experienced a return of previous symptoms, which were again relieved upon resuming trasentine medication.

Two patients who had previously received pyridoxine hydrochloride (vitamin B_6) without benefit obtained complete relief with trasentine. There was only one complaint relative to the administration of trasentine, namely that the tablets were "too sweet." The patient continued taking the drug, however, and obtained relief.

The pain complained of by some of the patients was described in each instance as "griping," which conforms to Kallet's (10) statement that "much of the tenesmus is due to sphincter spasm." In all of these cases moderate to complete relief was obtained.

In those patients who exhibited considerable apprehension, the use of trasentine-phenobarbital was of considerable value, in accord with the well recognized fact that

TABLE I: EFFECT OF TRASENTINE AND TRASENTINE-PHENOBARBITAL IN RADIATION SICKNESS

	Diagnosis	Symptoms	Treatment and Dose	Relief
1.	Carcinoma Cervix	Pain ++ Diarrhea +	T 3 × 4	+++ +++
2.	Carcinoma Cervix	Diarrhea +	T 2 × 3	+++
3.	Carcinoma Esophagus	Nausea ++	T 2 × 3	+++
4.	Carcinoma Cervix	Diarrhea +++	T 2 × 4	+++
5.	Carcinoma Cervix	Pain ++	T 2 × 3	+++
6.	Carcinoma Cervix	Diarrhea +++	T 3 × 3	0
7.	Carcinoma Cervix	Nausea ++	T 2 × 3	+++
8.	Carcinoma Cervix	Diarrhea +++	T 3 × 3	+++
9.	Carcinoma Cervix	Nausea ++ Diarrhea ++	T 2 × 3	+++ +++
10.	Carcinoma Cervix	Nausea +	T 1 × 3	+++
11.	Carcinoma Cervix	Pain ++ Nausea ++	T 1 × 4	++ +++
12.	Carcinoma Cervix	Nausea ++ Diarrhea +	T 1 × 3	+++ +++
13.	Carcinoma Cervix	Nausea ++ Diarrhea +++	T 2 × 3	+ 0
14.	Carcinoma Cervix	Nausea ++ Diarrhea +	T 3 × 3	++ +++
15.	Carcinoma Cervix	Nausea ++ Diarrhea ++	T 3 × 3	+++ +++
16.	Carcinoma Cervix	Nausea ++ Diarrhea +	T 3 × 3	++ ++
17.	Carcinoma Cervix	Nausea ++ Diarrhea ++	T 3 × 3	+++ +
18.	Carcinoma Cervix	Nausea ++ Diarrhea ++	T 3 × 3	+++ +++
19.	Carcinoma Cervix	Diarrhea ++	T 3 × 4	+++
20.	Carcinoma Cervix	Pain ++ Diarrhea ++	T 3 × 4	+++ +++
21.	Carcinoma Cervix	Diarrhea ++	T 3 × 4	+++
22.	Carcinoma Cervix	Nausea ++	T 2 × 3	++
23.	Carcinoma Cervix	Nausea ++ Diarrhea ++	T 3 × 3	++ +
24.	Carcinoma Rectum	Diarrhea +++	T 3 × 4	0
25.	Carcinoma Cervix	Diarrhea ++	T 2 × 4	+++
26.	Carcinoma Cervix	Nausea ++ Diarrhea ++	T 2 × 4	++ ++
27.	Carcinoma Cervix	Nausea ++	TP 1 × 4	+++
28.	Carcinoma Colon	Nausea +++	TP 2 × 3	++
29.	Carcinoma Cervix	Nausea ++ Pain ++	TP 2 × 3	+++ ++
30.	Marie-Strümpell Arthritis	Nausea ++	TP 1 × 4	+++
31.	Carcinoma Rectum	Nausea +++	TP 2 × 3	+++
32.	Marie-Strümpell Arthritis	Nausea ++	TP 2 × 3	0
33.	Carcinoma Cervix	Nausea ++	TP 2 × 3	+++

TABLE I: EFFECT OF TRASENTINE AND TRASENTINE-PHENOBARBITOL IN RADIATION SICKNESS (*Continued*)

Diagnosis	Symptoms	Treatment and Dose	Relief
34. Carcinoma Breast	Nausea ++	TP 2 × 3	+++
35. Carcinoma Cervix	Nausea +	TP 2 × 3	+++
36. Carcinoma Cervix	Nausea +++	TP 2 × 3	+++
37. Carcinoma Cervix	Nausea ++ Diarrhea +	TP 2 × 3	+++ ++
38. Carcinoma Cervix	Nausea ++	TP 2 × 3	0
39. Carcinoma Cervix	Nausea ++ Diarrhea +	TP 2 × 3	+ +
40. Carcinoma Cervix	Nausea ++ Diarrhea ++	TP 2 × 3	++ ++
41. Lymphosarcoma	Nausea ++	TP 2 × 3	+++
42. Carcinoma Stomach	Nausea +++	TP 2 × 3	++
43. Carcinoma Rectum	Nausea +	TP 2 × 3	++
44. Carcinoma Ovary	Nausea ++ "Nervousness" ++	TP 2 × 3	+++ +++
45. Hodgkin's Disease	Nausea ++	TP 2 × 3	+++
46. Carcinoma Cervix	Nausea ++	TP 2 × 3	+++
47. Carcinoma Cervix	Nausea ++	TP 2 × 3	++
48. Carcinoma Rectum	Nausea +++	TP 2 × 3	+++
49. Carcinoma Cervix	Nausea +	TP 1 × 3	++
50. Carcinoma Breast	Nausea ++	TP 1 × 4	+
51. Hodgkin's Disease	Nausea ++	TP 1 × 4	+++
52. Undiffer. Carcinoma	Nausea +	TP 1 × 4	+++
53. Carcinoma Cervix	Diarrhea ++	TP 1 × 4	0
54. Carcinoma Cervix	Nausea ++ Diarrhea ++	TP 1 × 4	+++
55. Carcinoma Cervix	Nausea +	TP 1 × 3	+++
56. Marie-Strümpell Arthritis	Nausea +	TP 1 × 3	+++
57. Hodgkin's Disease	Nausea ++	TP 1 × 4	+++
58. Carcinoma Cervix	Nausea +++	TP 1 × 4	+++
59. Carcinoma Cervix	Nausea + Diarrhea ++	TP 1 × 4, T 2 × 3	+ +++
60. Carcinoma Cervix	Nausea +++ Diarrhea ++	TP 1 × 3, T 2 × 3	+++ +++
61. Carcinoma Cervix	Nausea ++ Diarrhea ++	TP 1 × 3, T 3 × 3	++ ++
62. Carcinoma Cervix	Nausea + Diarrhea ++	TP 2 × 3, T 1 × 3	+++ +++
63. Carcinoma Cervix	Pain + Diarrhea ++	TP 1 × 4, T 2 × 4	+++ +++
64. Carcinoma Cervix	Nausea ++ Diarrhea +	TP 1 × 3, T 3 × 4	+++ +++
65. Carcinoma Cervix	Nausea +++ Diarrhea +++	TP 1 × 3, T 2 × 3	+++ 0

T. Trasentine. TP. Trasentine-phenobarbital compound. 1 × 4. One tablet four times a day. 2 × 3. Two tablets three times a day, etc. +. mild or slight. ++. moderate. +++. severe (or complete relief). 0. No relief.

a sedative is of benefit in some cases of radiation sickness. The combination of trasentine and phenobarbital in a single tablet is of advantage, also, in that it eliminates the necessity of two different medications.

We do not as yet know what, if any, influence trasentine may have upon the altered physiology of the intestinal tract, but studies of this problem are contemplated.

SUMMARY AND CONCLUSIONS

From data obtained up to the present time, we believe that trasentine and trasentine-phenobarbital are valuable adjuncts in the treatment of radiation sickness. No toxic symptoms were noted among the patients thus treated even though some received almost 1,000 mg. of trasentine per day (three 75 mg. tablets 4 times a day).

Trasentine seems to depress the appetite to some degree, but this factor is obviously difficult to evaluate in the type of patient under consideration. Conversely, some patients volunteered that their appetites were better after receiving trasentine. This may well be due to the relief of such disturbing symptoms as nausea and diarrhea.

NOTE: The trasentine and trasentine-phenobarbital used in this study were supplied through the courtesy of Ciba Pharmaceutical Products, Inc., Summit, N. J.

Duke University School of Medicine
Durham, N. C.

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A Simple, Accurate Fluoroscopic Method of Foreign Body Localization[1]

LT. COL. H. W. JAMISON, M.C., A.U.S.

THE METHOD OF foreign body localization herein described has proved in practice to be highly accurate and rapidly and easily accomplished. It has the added advantage of requiring no special apparatus. It is applicable to any fluoroscopic unit having some provision for fixing the distance from fluoroscopic screen to table top.

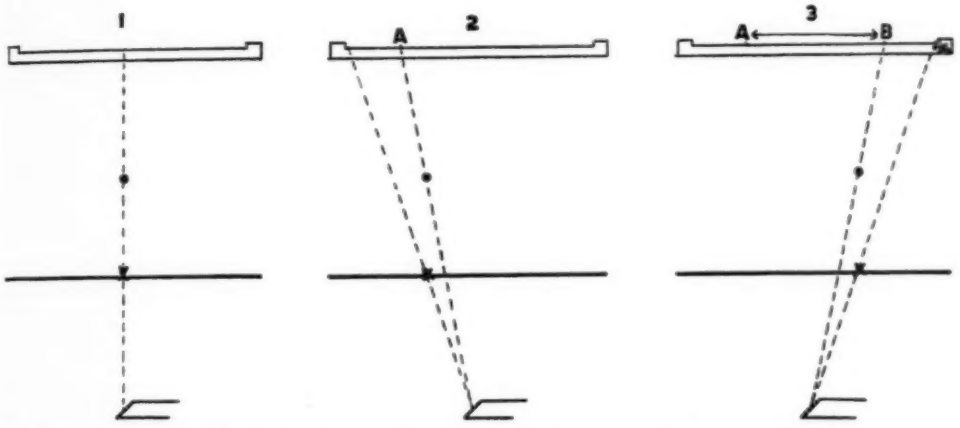

Fig. 1. Images of foreign body and skin marker superimposed at center of fluoroscopic screen.
Fig. 2. Fluoroscopic screen shifted cranially until the image of the skin marker coincides with the margin of the screen. The position of the foreign body image is indicated on the screen by A.
Fig. 3. Fluoroscopic screen shifted caudally until the image of the skin marker coincides with the opposite margin of the screen. The new position of the foreign body image is indicated on the screen by B. The depth of foreign body (from skin marker) is inversely proportional to distance AB.

The patient is placed on the fluoroscopic table. The fluoroscopic screen is fixed at a predetermined distance from the table top, and its illumination is limited to a narrow beam centered directly over the foreign body to be localized. A lead cross or other metallic marker is affixed by adhesive tape to the patient's skin on the dependent side in such a position that the projected images of the foreign body and cross are superimposed. The fluoroscopic beam is then adjusted to a narrow slit parallel to the long axis of the body, extending across the full diameter of the screen. The screen is moved cranially until the center of the image of the skin marker coincides with the edge of the screen. Then, with a soft wax pencil, a mark is made on the screen at the center of the projected foreign body image. The screen is next shifted caudally until the shadow of the skin marker coincides with the opposite margin of the screen. A second mark is made with the wax pencil at the new position of the foreign body image. The distance between wax marks on the fluoroscopic screen is then measured, and, by reference to a calibration chart, the depth of the foreign body can be read off directly. The point of attachment of the lead marker is indicated on the skin by wax pencil at the time the marker is removed. The position of the foreign body can then be reported as being "—— cm. deep to the mark on the skin."

Localization depends on the relative shift of the projected foreign body image

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on the fluoroscopic screen as compared with that of the lead skin marker, which, being attached to the dependent portion of the patient's body, is at table-top level. By using the diameter of the fluoroscopic screen as a measure, the skin marker image shift is kept constant. The screen-table top and focal-screen distances are also fixed. Thus, the only variable factor is the shift of the foreign body image, which, in turn, is proportional to depth of the foreign body in the tissues.

A calibration chart indicating depth of foreign body in relation to foreign body image shift can be constructed mathematically, given focal-table top, focal-foreign body, and focal-screen distances. More simply, calibration can be made by using a phantom, such as a discarded film box top, to which appropriate lead numbers are fixed at 1 cm. intervals from fluoroscopic table.

This method of localization has several advantages: It is based on triangulation using large dimensional relations and hence is highly accurate; it requires no special apparatus other than some method for fixing table top-fluoroscopic screen distance and is therefore applicable to almost all fluoroscopic units; it requires only a few seconds for its accomplishment.

2009 Wilshire Blvd.
Los Angeles 5
Calif.

Medical and Hospital Insurance Problems[1]

ARTHUR W. ERSKINE, M.D.
Cedar Rapids, Iowa

DOCTORS WHO discuss subjects like the one assigned to me must be careful not to use the words "socialism" or "socialistic" as epithets. If by the philosophy of socialism we mean that the strong should help to bear the burdens of the weak, doctors are the oldest and almost the only real socialists. Since the dawn of civilization, long before Karl Marx expounded his doctrines, they have adjusted their fees to the ability of their patients to pay in an effort to see that the rich shall meet the expense of supplying medical service to the poor. As a class, they believe in the general principles of insurance, that thrift is a virtue, and that the wise will budget against heavy unforeseen expenditures. I have asked many of my own colleagues how they would like to have their county medical society practice as a guild, receiving two dollars per month for each man, woman, and child in the county, this money being distributed according to the ability, energy, and popularity of the members. The great majority of those questioned appeared to believe that under an *ideal* administration of such a plan, with overhead expense cut to a minimum, with no duplication of equipment or labor, and with the time-consuming amenities incidental to competitive private practice abolished, they would find their incomes greater, their work easier, their responsibilities lighter, and their leisure time longer. Doctors do not object to having their fees paid. They support workmen's compensation laws in the various states in spite of the fact that these laws provide professional services under a compulsory insurance plan.

Why, then, do doctors invariably view with suspicion any proposal to supply medical services through such agencies as the Blue Cross, voluntary medical insurance plans, or compulsory insurance plans as proposed in the Wagner-Murray-Dingell bill. I think the answer is simple. It is not because these proposals are socialistic. What is feared is not socialization but regimentation. We fear, and we have good reason to fear, the introduction of a third party between ourselves and our patients. This third party would have the power, because he would hold the purse-strings, to say what patients should be treated, how long, and in what manner, though his only qualification for his job might be that he is a good, faithful wheel horse of his party. We have reason to fear regimentation because of our experience with the care of the indigent sick during the depression. When social workers or overseers of the poor had to contend not only with social problems, but also those which could be solved only by professionals, the whole administration of medical relief was unsatisfactory to everyone concerned. Some progress in efficiency, economy, and the promotion of the welfare of the patients began to be made only when social and medical problems were completely divorced, the former being solved by trained social workers, the latter by medical men.

In its opposition to regimentation and political domination, three courses lie open to the organized medical profession. We might listen to those, some of them sitting in the seats of the mighty, who believe that every proposal for change should be opposed. If the entire membership should stand with a united front, such opposition might be successful, but it is apparent that unanimity of opinion among all the members of a profession like ours is impossible, although we can expect, and can demand, unanimous action based upon the will of

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the majority. But there are many doctors who believe changes in our methods of furnishing medical care are necessary and desirable; there are many more who are convinced that changes are inevitable and that it is our function to see that they are wise changes. The second course of action is to abandon completely those principles to which all honorable doctors subscribe, and to the support of which they dedicate their lives, and supinely accept all proposals, in spite of the fact that we know some of them would turn back the practice of medicine to medieval times. One course would result in a program being forced upon us which would eventually reduce the standards of medical practice to the level of those in Central Europe. The other would result in a hopeless division within our own ranks.

Organized American physicians must follow a third course, approaching their task in a spirit of conciliation and compromise. In spite of what our so-called spokesmen say, those who deal directly with patients know that the problem of supplying adequate medical service to the underprivileged does exist. They know that many men and women fail to get needed medical service because "to beg they are ashamed." This is our problem and that of the people. Even now, it is not too late for us to solve it, if we choose, by promoting hospital insurance, insurance against catastrophic illness, and by controlled experimentation with plans for prepaid medical service to members of the low-income group by medical societies. We must differentiate sharply between mass medical service under political domination, a thing we all hate and fear, and mass medical service administered and controlled by medical organizations. The physician must remain "the master in the house of medicine." He must be kept free from political control. If these things can be done, and I think they can, we have no more reason to fear changes in methods of practice than we have cause to regret the economic changes we have already seen in our own professional lifetime.

The problems of radiologists, as they are related to hospital and medical insurance, are more complex than those of the general medical profession. The nature of their calling compels radiologists not only to practice an art, but also to conduct a business. X-ray apparatus is expensive, and so much capital is required that many a young radiologist either prefers or is driven by necessity to accept a salaried position instead of establishing his own practice. Overhead is high. If only half our patients paid us, or if our average fees were reduced one-half without an increase in volume, most of us would soon be bankrupt. The American Hospital Association, the Blue Cross, and most of its member hospitals appear to be determined to include x-ray service as an integral part of hospitalization in spite of the opposition of the organized medical profession. They want to be allowed to buy professional x-ray service at wholesale and to sell it at retail. Finally, many doctors, perhaps a majority of them, would like x-ray examinations to be free to their patients and paid for by insurance or by taxation, as Wassermann tests are paid for now. The most bitter opponents of national compulsory health insurance bills, including the president of the A. M. A. in 1943, seem to agree that the government can properly establish "diagnostic centers" wherever they are needed. Those who are thoughtful and observant are forced to the conclusion that radiologists are apt to follow the pathologists to the altar as a sacrifice to "conciliation and compromise."

On the other hand, we are not altogether helpless. We are a small, compact group, knit closely together in associations, and we have the American College of Radiology to protect our economic interests. We are trained to observe. We have seen the fate of our colleagues, the pathologists; we can see what may be in store for us. We are an essential group; the hospitals and the medical profession need us, our cooperation, and support, more than we need them. While the great majority of radiologists would prefer to continue to practice

as specialists, they need not do so. If they should be abandoned by their professional brethren, many radiologists could do fairly well in gastroenterology or internal medicine, most of them could treat fractures adequately, and many could qualify in dermatology and other specialties.

In our dealings with hospitals and with hospital insurance plans, it is vitally important that we continue to insist upon a sharp differentiation between hospital services and professional services. There are three compromises which we can safely accept:

1. Radiology can be excluded from Blue Cross and included in the medical service plan offered to Blue Cross subscribers. This is both the most logical and desirable compromise.

2. The medical service plan can issue a supplemental contract paying cash benefits for radiology, pathology, and anesthesiology, to be sold with the Blue Cross contract to subscribers.

3. Blue Cross can itself pay cash benefits for medical services, thus actually separating them from hospital care. This compromise was adopted in the states of Washington and Iowa.

At the 1943 meeting of the Iowa State Medical Society, a reference committee was appointed to study the proposal of the Blue Cross to expand its benefits to include x-rays, anesthesia, and certain other professional services *as a part of hospital service.* The hearings of this committee were attended by representatives of the Iowa X-Ray Club and Mr. Mac Cahal of the College of Radiology. The committee recommended that services which, legally and ethically, can be rendered only by licensed physicians be excluded from the hospital service plan. The recommendation was adopted by the House of Delegates. Late in 1943, however, the Executive Council of the State Society reversed the decision of the House of Delegates and approved the Blue Cross proposal, going so far as to say that fees for such professional services should be paid by the insurance company to the hospitals as agents of the doctors. Incidentally, the transcript of the minutes of this meeting shows very clearly that many members of the Executive Council failed to grasp the implications of the proposal.

At its 1944 meeting, the Iowa X-Ray Club appointed a committee to meet with a committee from the directors of the Blue Cross to try to arrive at a satisfactory compromise. These men were very sympathetic to our position which, briefly, was that the insurance company could properly undertake to furnish hospital service, but not professional service over which it had no control. We did not, however, object to the insurance company undertaking to pay for such professional services, provided that they were clearly separated from hospital services. But the Board of Directors refused to accept the recommendation of its own committee. Shortly afterward the secretary of the Iowa X-Ray Club wrote a letter to members of the Executive Council of the medical society and to some of the directors of the Blue Cross, in which he said that, in spite of our efforts at compromise, the Blue Cross was apparently determined to practise medicine, and that since we have a pretty good medical practice act in Iowa, perhaps the quickest, cheapest, and easiest way of settling the controversy would be for the Iowa X-Ray Club, through one of its members, supported and financed by the medical society, to file a suit against the Blue Cross or one of its member hospitals, asking for an injunction to restrain the company or its member hospitals from practising medicine. This was not a threat; it was just a suggestion. At the next meeting of the Directors of the Blue Cross, they adopted a new contract, which from our standpoint is satisfactory because it contains these two paragraphs:

"The Service Corporation further agrees to make the following credit allowance toward the physician's fee for the professional medical services within the limits as outlined below . . . . .

"Professional medical services as used in this contract refers to services which legally and ethically can be performed only by a licensed physician but for which the Service Corporation agrees to pay. The Service Corporation cannot provide, and does

not undertake to furnish some services, because it can exercise no direction or control over any physician or surgeon."

What else can we do to ensure that radiology shall continue to endure as a specialty? Fortunately, we are well aware of the dangers that threaten it, and we can face them unitedly, realistically, and with some confidence in the weapons ready to our hands. Radiologists can properly use any legitimate methods of enhancing their reputations among the public. We often receive patients who have spent some time in great metropolitan hospitals. These patients all know the names of their attending physicians or the surgeons who operated upon them. None of them knows the name of the roentgenologist who examined him and probably made the diagnosis. Practically all of them believe that "a girl" made the x-ray examinations. Forty per cent of the qualified radiologists of the United States (50 per cent if the four largest cities are excluded) practise in cities of less than 100,000 population. The radiologists in the smaller cities can all spend a minute or two talking to each of their patients and showing some interest in their welfare. Those who work in great metropolitan centers, if they are really consultants, might well do the same thing, even though it might require them to make rounds.

Radiologists who conduct their own practices and work in hospitals in competition with themselves have a stronger position in their relationship to hospitals and insurance companies than those on salaries. I suggest that we should plan *now* to absorb in partnerships as many young radiologists as we can on their release from the Armed Services. This will not only lessen the number of radiologists without much capital who might be tempted to accept salaried positions for the sake of security; it will also tend to reduce competition, with its attendant wasteful duplication of equipment and overhead. Modern radiology now covers such a broad field that no individual can hope to learn it all. Important decisions must be made, some of them,

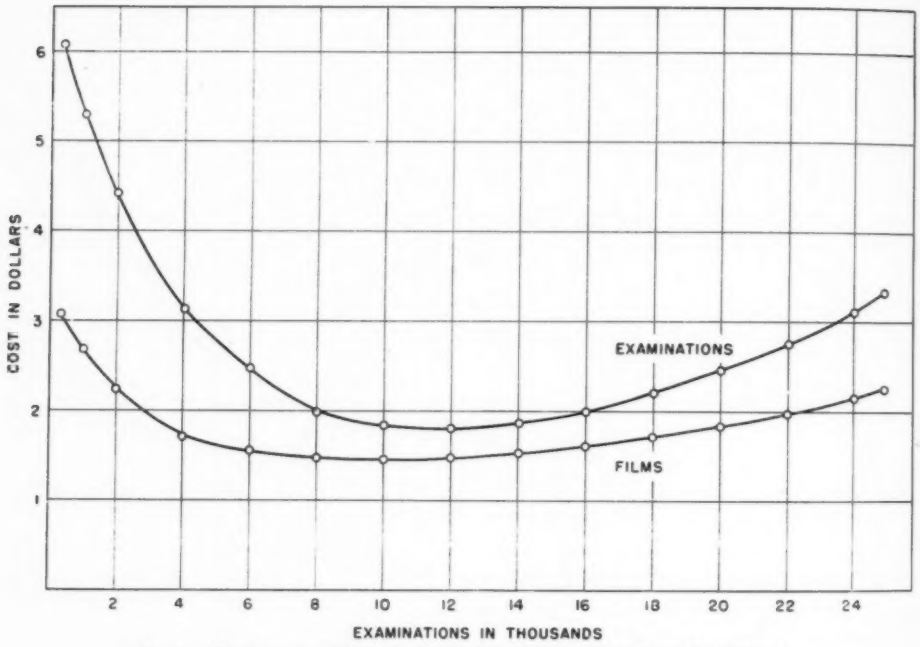

Fig. 1. Cost per examination and per film in Iowa plotted against volume.

literally, between life and death. It is comforting to be able to share the heavy burdens of responsibility that we all must carry. Partnerships and similar combinations also have the tremendous advantage of utilizing the skill, training, strength, endurance, and enthusiasm of youth immediately and of avoiding a long, difficult, discouraging struggle to build up a practice.

Finally, the high cost of sickness is largely responsible for much of the present agitation for radical innovations in our system of medical care. Perhaps we should seriously consider the possibility of reducing the cost of x-ray diagnosis by any means consistent with good service. In the past year a committee of the Iowa X-Ray Club has made a study of the cost of various x-ray diagnostic procedures, including overhead and depreciation at various levels of volume, and is making estimates of the degree to which fees could be reduced without reduction of net income, if collection expense were eliminated and if volume were multiplied one and one-half, two, or three times. The committee was appointed because most of us would prefer a fee-for-service basis of compensation to a salary, because reduction of fees might well be the best way to meet the threat of tax-supported "diagnostic centers," and also because even the Wagner-Murray-Dingell bill provides for payment on a fee basis. The committee limited its investigation to learning the cost per examination and per film. These costs, and especially their dependence on volume, are shown graphically in Figure 1. If the Wagner-Murray-Dingell bill or some modification of it should be passed, it would be to our advantage to have some knowledge of average costs and possible reductions of fees. Obviously, they would not be the same in all communities, and similar investigations and estimates by state and national radiological societies might be useful.

326 Higley Bldg.
Cedar Rapids, Iowa

EDITORIAL

To the Members of the Radiological Society of North America

The Thirty-second Annual Meeting of the Radiological Society of North America will be held at the Palmer House in Chicago, the first week in December. Sessions begin on Monday, December 2nd, and extend through Friday, December 6th.

Since this is the first full-scale meeting that the Society has held since 1941, it is expected that there will be a large attendance. The scientific exhibits and refresher courses, and the program (which appears elsewhere in this issue) will, I believe, repay everyone who attends. The various members of the Program Committee have worked untiringly to secure a series of excellent presentations, and the Committees in charge of the refresher courses and the exhibits have been no less diligent in the preparation of these features.

It is apparent to every radiologist that the successful harnessing of atomic energy opens up vast fields of new knowledge. Artificial radioactivity and the use of various radioactive isotopes in diagnostic and therapeutic medicine will be of the greatest importance in the future, and these subjects are of particular interest to radiologists, since their basic training makes them peculiarly fitted to employ these methods intelligently.

Because of this, the Program Committee has devoted the entire opening day of the Annual Meeting to the Plutonium Project. Nine papers will be presented by some of the most distinguished scientists in America. The entire Society owes a debt of gratitude to these men who have willingly and cheerfully undertaken this project.

LOWELL S. GOIN, M.D.
President

The Radiologist's Report

The radiologist's share in shaping the type of medicine practised in a community, clinic, or hospital is considerable. His report, in many cases, furnishes the final evidence for diagnosis; upon it treatment is based, and it may be the deciding factor in the settlement of a claim for compensation or insurance. It behooves him, therefore, to use a language which is correct and yet understandable without being stilted; to be concise but explicit. The accuracy of his words and descriptions and his regard for the finer shades of meaning will be taken as a measure of his professional ability. In no other specialty is the slightest misuse of a word or phrase so likely to reflect upon the writer. The radiologist, therefore, should clean his verbal house.

Examples come readily to mind. Many radiologists, for instance, speak of "getting accommodated" or "dilated" in the fluoroscopic room, whereas what actually takes place is the adaptation of the retina to the dark. It is the pupil that is dilated and the lens that is accommodated, the former reacting almost instantaneously to changes in the amount of light, the latter changing its thickness so as to keep the image in focus on the retina.

Another type of misuse is exemplified by the term "prespondylolisthesis" an unfortunate mixture of Latin and Greek

which is used to designate a defect in the neural arch without anterior slipping of the vertebral body. While the word describes the condition fairly well, it carries the implication that the vertebra *will* slip. Either spondyloschisis (from the Greek words for vertebra and fissure) or spondylolysis (from the Greek for vertebra and dissolution) is a better term for a neural arch defect when no slipping has occurred. Or, if one objects to the rather bookish Greek words, he may employ the English description instead.

This example has a special application in reporting the facts as observed on the x-ray film and differentiating between these and the opinions or implications which they are believed to justify. The radiologist should report what he sees on the film, and then draw a logical conclusion; if that is clear, nothing further need be written. On the other hand, if it is not clear or if his opinion is at variance with the shadows described, he should state this unequivocally. In this way, implied meanings are unveiled and clarified. The word "prespondylolisthesis" implies, as well as describes, and to the uninitiated (and/or the insurance company) the implication may be that, because there is the possibility of a slip, there will inevitably be a slip. The term is indeed a dangerous one, and the sooner it is discarded the better.

Other examples of unjustifiable implication are to be found in reports of chest examinations. The most frequent of these is the unqualified interpretation of certain densities in the lungs as "tuberculosis in the apex." This implies that the roentgenogram is able to furnish enough differential evidence to distinguish the exact cause of the pulmonary shadows. Actually a specific bacterial diagnosis is not possible on the basis of the roentgen findings alone, though the statistical chances are high that densities found in a particular location and of a particular character are tuberculous.

Another regrettable practice is the description of masses or densities as "the size of a walnut," or a grapefruit, or some other more or less familiar object. If the radiologist would have his confreres respect his words, he should discard such unprecise estimates and accurately measure and report dimensions in centimeters or inches (and in the use of these units he should be consistent). This also holds for fractures. When separation of fragments is being considered, exact measurements are of great help. When displacements of fragments in the shaft of a bone are involved, the percentage of offset is even more useful, since here the relative displacement is more important than the absolute.

Pressure is frequently brought to bear upon the radiologist to turn his roentgenographic findings into a clinical diagnosis, as "mucous colitis" or "chronic appendicitis." To interpret such a roentgenographic finding as slight irritability of the colon as "mucous colitis," or slight stasis of barium in the appendix as "chronic appendicitis," endangers the scientific approach that it is the radiologist's privilege and obligation to impart to medicine.

SYDNEY F. THOMAS, M.D.

Photo by J. Anthony Bill

Ellis R. Bader, M.D.

1888–1946

On June 16, 1946, death came to Ellis Robert Bader of Cincinnati at the age of fifty-eight. For twenty-five years he had been associated with the late Dr. William M. Doughty, whose death preceded his by exactly twenty-six months. For over a year and a half Dr. Bader had suffered from chronic myelogenous leukemia, but the immediate cause of his passing was a cerebral thrombosis. He was well aware of what was ahead of him during these twenty months and he faced the inevitable with remarkable courage.

To those who knew Dr. Bader, certainty of pur-

pose characterized his life. He was unswervingly devoted to his home and family and after these came his professional career, which he followed with fixed and definite purpose.

Dr. Bader was graduated from the University of Cincinnati College of Medicine in 1913. He enlisted in the British Army and later transferred to the United States Army in World War I. During his professional life, he held many important positions. He had been radiologist at Bethesda and Deaconess Hospitals in Cincinnati and at St. Elizabeth Hospital in Covington, Kentucky. At the time of his death, he was radiologist at Christ Hospital in Cincinnati. He was also on the consulting staff at Children's Hospital. For many years he had been Assistant Professor of Radiology at the University of Cincinnati College of Medicine. Always active in organized medicine, he had served the Cincinnati Academy of Medicine and the Ohio State Radiological Society as secretary. He had been a member of many important committees of the local and state medical societies. In 1937, he was first vice-president of the Radiological Society of North America and, prior to that, he had been Counselor for Southern Ohio for many years. At the time of his death, he was the second vice-president of the American Roentgen-Ray Society and Councilor of the American College of Radiology for Southern Ohio. He is survived by his wife, Dorothy Adams Bader, a daughter, Harriet Bader Wilks, who is a registered nurse, and a son, Robert, who is a medical student.

Dr. Bader had an unusually wide circle of friends and acquaintances in the Cincinnati area. He was known for his honesty and integrity and had built an enviable reputation as a radiologist. In his life he reflected only credit on the practice of medicine in general and on the specialty of radiology in particular. The Radiological Society of North America has lost a valuable member and his associates a devoted friend.

HAROLD G. REINEKE, M.D.

Clark Anson Wilcox, M.D.

1890–1946

Dr. Clark A. Wilcox, of Wichita Falls, Texas, a member of the Radiological Society of North America since 1920, died on April 4, 1946, of aplastic anemia.

Dr. Wilcox was born in Scottsville, N. Y., in 1890, was graduated from the University of Michigan, and received his degree in medicine from the New York Homeopathic College and Flower Hospital. In 1917 he was commissioned a first lieutenant in the Medical Reserve Corps of the Army. After several years in the Reserve, he accepted the commission of captain in the Medical Corps of the U. S. Army, later resigning to enter civilian practice in Wichita Falls. He was made a major in the Reserve Corps in 1924 and was promoted to lieutenant colonel in 1931. From 1923 until his death Dr. Wilcox was radiologist with the Wichita Falls Clinic and Clinic-Hospital.

Dr. Wilcox was a diplomate of the American Board of Radiology, a fellow of the American College of Radiology, and a fellow of the American College of Physicians. He is survived by his wife, Ruth Gracelon Wilcox, and two daughters, Miss Allene Wilcox and Mrs. J. R. Kolb, Jr.

RADIOLOGICAL SOCIETY OF NORTH AMERICA

THIRTY-SECOND ANNUAL MEETING, PALMER HOUSE, CHICAGO
DEC. 2–DEC. 6, 1946

Monday, December 2

GENERAL SESSION: 10:30 A.M.

CALL TO ORDER

LOWELL S. GOIN, M.D., President

Los Angeles, Calif.

ADDRESS OF WELCOME

ROBERT S. BERGHOFF, M.D.

President of Illinois State Medical Society

PRESIDENTIAL ADDRESS

RADIOLOGY AND THE FUTURE

LOWELL S. GOIN, M.D.

THE PLUTONIUM PROJECT

Chairman: Austin M. Brues, M.D.

Director, Biology Division, Argonne National Laboratory, and Associate Professor of Medicine, University of Chicago

1. Some Biological Effects of Neutrons, RAYMOND E. ZIRKLE, PH.D., Director, Institute of Radiology and Biophysics, and Professor of Botany, University of Chicago.
2. Biological Studies in the Tolerance Range, EGON LORENZ, PH.D., Principal Biophysicist, National Cancer Institute, Bethesda, Md.

COUNSELLORS' LUNCHEON

GENERAL SESSION: 2:00 P.M.

THE PLUTONIUM PROJECT *(Continued)*

3. An Evaluation of Blood Changes from Radiation Exposure Near the Tolerance Level, LEON O. JACOBSON, M.D., Assistant Professor of Medicine, University of Chicago.
4. The Metabolism of Fission Products, JOSEPH B. HAMILTON, M.D., Associate Professor of Medical Physics, University of California.
5. Some Physiological Effects of Radiation, C. LADD PROSSER, PH.D., Associate Professor of Physiology, University of Illinois.
6. Effects of Beta Rays on the Whole Animal, JOHN R. RAPER, PH.D., Assistant Professor of Botany, University of Chicago.
7. Histological Changes Following Radiation Exposures, WILLIAM BLOOM, M.D., Professor of Anatomy, University of Chicago.
8. Tumor Production by Radioactive Materials, AUSTIN M. BRUES, M.D.
9. Biologic Effects of Pile Radiations, PAUL S. HENSHAW, PH.D., Biologist, Monsanto Chemical Company, Clinton Laboratories, Knoxville, Tenn.

MEMBERSHIP DINNER AND EXECUTIVE SESSION: 7:00 P.M.

Tuesday, December 3

GENERAL SESSION: 10:30 A.M.

1. Quantitative Data on the Characteristics of Commercial X-Ray Films and Screens, RUSSELL H. MORGAN, M.D., Chicago, Ill.
   Discussion: W. EDWARD CHAMBERLAIN, M.D., Philadelphia, Penna.
2. Isotopes in Medical Investigation and Therapy, JOHN H. LAWRENCE, M.D., Berkeley, Calif.
3. Deposition of Radioactive Iodine in the Normal and Malfunctioning Thyroid Gland in Children, EDITH H. QUIMBY, SC.D., AND DONOVAN MCCUNE, M.D., New York, N. Y.
4. Application of Radioactive Isotopes to a Study of Radiation Effects in Cells, MARTIN D. KAMEN, PH.D., St. Louis, Mo.
   Discussion (of papers 2, 3, 4): W. EDWARD CHAMBERLAIN, M.D., Philadelphia, Penna.

EXECUTIVE SESSION 2:00 P.M.

DIAGNOSTIC SESSION: 2:30 P.M.

1. Catheterization of the Heart; Indications, Errors, and Technic, MERRILL C. SOSMAN, M.D., Boston, Mass.
2. Catheterization of the Heart; Results, Interpretations, and Value, L. F. DEXTER, M.D., Boston, Mass. (by invitation).
   Discussion (of papers 1 and 2): WENDELL G. SCOTT, M.D., St. Louis, Mo.
3. Differential Diagnosis of Retrocardiac Shadows, STANLEY S. NEMEC, M.D., St. Louis, Mo. (by invitation).
   Discussion: LEROY SANTE, M.D., St. Louis, Mo.
4. Calcification of the Cerebral Basal Ganglia and Its Roentgenological Significance, JOHN D. CAMP, M.D., Rochester, Minn.
   Discussion.

5. X-Ray Signs of Altered Alimentary Function Following Autonomic Blockade with Tetra Ethyl Ammonia. JOHN F. HOLT, M.D., RICHARD H. LYONS, M.D., GORDON K. MOE, M.D., AND FRED J. HODGES, M.D., Ann Arbor, Mich.

THERAPY SESSION: 2:30 P.M.

SYMPOSIUM ON CARCINOMA OF THE UTERINE FUNDUS AND CERVIX

Chairman: Edw. C. Ernst, M.D.
St. Louis, Mo.

1. Pathological Aspects, ROBERT MOORE, M.D., Dean and Professor of Pathology, Washington University, St. Louis, Mo.
2. Clinical Phases of the Cervical Cancer Problem, A. N. ARNESON, M.D., Barnard Hospital, St. Louis, Mo.
3. Some Aspects of Radium Therapy, H. H. BOWING, M.D., Mayo Clinic, Rochester, Minn.
4. Transvaginal Radiation Methods, JUAN A. DEL REGATO, Missouri State Cancer Hospital, Columbia, Mo.
5. The Surgical Aspects of Uterine Carcinoma, JOHN BREWER, M.D., Chicago, Ill.
6. Five Year End-Results in the Treatment of Carcinoma of the Uterine Cervix, EDWARD L. JENKINSON, Chicago, Ill.

THE CARMAN LECTURE: 8:00 P.M.

QUALITY OF RADIATION IN THERAPY

ROBERT R. NEWELL, M.D.
Professor of Radiology, Stanford University
San Francisco, Calif.

Wednesday, December 4

GENERAL SESSION: 10:30 A.M.

1. Filter Value Inherent in Very High Voltage Radiation Therapy Equipment, E. DALE TROUT AND Z. J. ATLEE, Chicago, Ill.
2. Effects of Estradiol Benzoate on Bone Formation and Blood Calcium Levels in the Cockerel and Their Modification by Bleeding, KURT SALOMON, M.D., PH.D., BEVERLY WESCOTT GABRIO, A.B., EDWARD REINHARD, M.D., AND RUTH SILBERBERG, M.D., St. Louis, Mo.
   Discussion.
3. Experimental Clostridial Infections in Dogs, ANDREW H. DOWDY, M.D., Rochester, N. Y.
   Discussion: ROBERT P. BARDEN, M.D., Philadelphia, Penna.
4. Further Studies on the Relation between Radiation Effects, Cell Viability and Induced Resistance to Malignant Growth: IV. Effects of Roentgen Rays on Mammary Tumors Autogenous to Inbred Strains of Mice (dbrB and C3H), ANNA GOLDFEDER, D.Sc., M.U.C., New York, N. Y.
   Discussion: G. FAILLA, PH.D., New York, N. Y.

DIAGNOSTIC SESSION: 2:00 P.M.

1. Problems in the Diagnosis of Cancer of the Colon, KENNETH S. DAVIS, M.D., AND WILLIAM DANIELS, M.D., Los Angeles, Calif.
   Discussion.
2. Roentgen Ray Diagnosis of Pigmented Villonodular Synovitis and Synovial Sarcoma of the Knee Joint: Preliminary Report, RAYMOND W. LEWIS, M.D., New York, N. Y. (by invitation).
   Discussion: JACOB GERSHON-COHEN, M.D., Philadelphia, Penna.
3. The Adult Silent Chest, GEORGE M. LANDAU, M.D., AND HILDEGARD A. SCHORSCH, M.D., Chicago, Ill.
   Discussion: EARL E. BARTH, M.D., Chicago, Ill.
4. Giant-Cell Tumor of Bone, FRANKLIN B. BOGART, M.D., Chattanooga, Tenn., AND ALLISON E. IMLER, M.D., Birmingham, Ala.
   Discussion.
5. Appendiceal Coproliths, S. F. THOMAS, M.D., Palo Alto, Calif.
6. Cardio-Esophageal Relaxation as a Cause of Vomiting in Infants, EDW. B. D. NEUHAUSER, M.D., AND WM. BERENBERG, Boston, Mass.
   Discussion.

THERAPY SESSION: 2:00 P.M.

1. Giant Follicular Lymphadenopathy (Brill-Symmer's Disease), ERICH M. UHLMANN, M.D., Chicago, Ill.
   Discussion.
2. An Evaluation of Radiation Therapy of Carcinoma of the Skin, CLAYTON H. HALE, M.D., Syracuse, N. Y.
   Discussion.

SYMPOSIUM ON THE TREATMENT AND CARE OF ADVANCED CANCER PATIENTS

Chairman: H. H. Murphy, M.D.
Victoria, B. C.

3. Introduction, H. H. MURPHY, M.D.
4. Care of Patients with Pelvic Cancer, WM. E. COSTOLOW, M.D., Los Angeles, Calif.
5. Care of Patients with Breast Cancer, ARTHUR W. ERSKINE, M.D., Cedar Rapids, Iowa.
6. Care of Patients with Mouth and Throat Cancer, GRANT BECKSTRAND, M.D., Long Beach, Calif.
   Discussion (of papers 4, 5, 6): H. H. MURPHY, M.D.

Thursday, December 5

GENERAL SESSION: 10:30 A.M.

1. Medical, Biological, and Industrial Applications of Monochromatic Radiography and Microradiography, GEORGE L. CLARK, PH.D., D.Sc., Urbana, Ill. (by invitation).
   Discussion: KENNETH E. CORRIGAN, PH.D., Detroit, Mich.

2. High Intensity Radiation from Beryllium Window X-Ray Tubes, T. H. ROGERS, Springdale, Conn. (by invitation).
Discussion.
3. Development of the Betatron for Electron Therapy, L. S. SKAGGS, Chicago, Ill., G. M. ALMY, D. W. KERST, AND L. H. LANZL, Urbana, Ill. (by invitation), with Introduction by ERICH M. UHLMANN, M.D., Chicago, Ill.
Discussion.
4. Importance of Alveolar Pressure in the Diagnosis of Pulmonary Disease, DR. NILS WESTERMARK, St. Göran's Hospital, Stockholm, Sweden (by invitation).

DIAGNOSTIC SESSION: 2:30 P.M.

1. Deformities of the Greater Curvature of the Stomach, MAURICE FELDMAN, M.D., Baltimore, Md.
Discussion: KENNETH S. DAVIS, M.D., Los Angeles, Calif.
2. Erosive and Proliferative Changes in the Bones and the Soft Tissues of Paraplegics, NORMAN HEILBRUN, M.D., AND WM. G. KUHN, M.D., Buffalo, N. Y.
Discussion: PAUL C. SWENSON, M.D., Philadelphia, Penna.
3. Roentgenologic Aspect of Certain Lesions of Bone: Neurotropic or Infectious, JOHN R. HODGSON, M.D., DAVID G. PUGH, M.D., AND H. H. YOUNG, M.D., Rochester, Minn.
Discussion.
4. Roentgen Diagnosis of Localized Benign Metaphyseal Fibrous Lesions, LILIAN DONALDSON, M.D., Chicago, Ill.
Discussion: C. HOWARD HATCHER, M.D., Chicago, Ill.
5. Bagasse Disease of the Lungs—A Preliminary Report. DAVID V. LEMONE, M.D., Columbia, Mo., WENDELL G. SCOTT, M.D., SHERWOOD MOORE, M.D., and Capt. A. LINK KOVEN, U.S.P.H.S., St. Louis, Mo.
Discussion: Capt. A. LINK KOVEN, U.S.P.H.S.
6. Acute Obstruction of the Small Bowel—The Value of Roentgenological Examination in Early Diagnosis, JACK SPENCER, M.D., AND LANGDON T. THAXTER, M.D., Portland, Maine.
Discussion.

THERAPY SESSION: 2:30 P.M.

1. Further Observations on the Intra-Vaginal Roentgen Treatment of Cervical Cancer, W. WALTER WASSON, M.D., Denver, Col.
Discussion: ARTHUR W. ERSKINE, M.D., Cedar Rapids, Iowa.
2. Experience with Nasopharyngeal Carcinoma, VINCENT P. GRAHAM, M.D., AND RALPH R. MEYER, M.D., Chicago, Ill.
Discussion: FRED J. HODGES, M.D., Ann Arbor, Mich.

SYMPOSIUM ON LYMPHOBLASTOMA

Chairman: R. G. Willy, M.D.
Chicago, Ill.

3. Introduction, R. G. WILLY, M.D.
4. ANNA HAMANN, M.D., Chicago, Ill.
5. LEON JACOBSON, M.D., Chicago, Ill.
6. Therapeutic Use of Radioactive Isotopes, JOHN H. LAWRENCE, Berkeley, Calif. (by invitation).
Discussion.

ANNUAL BANQUET: 7:30 P.M.

Friday, December 6

GENERAL SESSION: 10:30 A.M.

1. Studies of Tetraiodophthalamidoethanol as a Medium for Gastro-Intestinal Visualization, GLENN E. JONES, WILLIAM E. CHALECKE, M.D., JOSEPH DEC, PH.D., JOHN SCHILLING, M.D., HAROLD D. ROBERTSON, GEORGE H. RAMSEY, M.D., AND WILLIAM H. STRAIN, PH.D., Rochester, N. Y. (by invitation).
Discussion.
2. Studies on Emulsions of Ethyl Iodophenylundecylate as Media for the Visualization of Body Cavities, WILLIAM E. CHALECKE, M.D., GLENN E. JONES, MURRAY P. GEORGE, M.D., EARLE B. MAHONEY, M.D., AND WILLIAM H. STRAIN, PH.D., Rochester, N. Y. (by invitation).
Discussion.
3. The Endocrine Factors in Pelvic Tumors, with a Discussion of the Papanicolaou Smear Method for Diagnosis, A. E. RAKOFF, M.D., Philadelphia, Penna. (by invitation).
Discussion: J. P. GREENHILL, M.D., Chicago, Ill.
4. Calcium, Phosphorus and Phosphatase as Aids in the Diagnosis of Bone Lesions. EDMUND FLINK, M.D., Minneapolis, Minn. (by invitation).

DIAGNOSTIC SESSION: 2:00 P.M.

1. Infantile Cortical Hyperostoses: A New Syndrome, JOHN CAFFEY, M.D., New York, N. Y.
Discussion: PAUL C. SWENSON, M.D., Philadelphia, Penna.

SYMPOSIUM ON DISEASES OF THE CHEST

Chairman: L. H. Garland, M.D.
San Francisco, Calif.

2. Reasons for the Common Anatomic Location of Pulmonary Tuberculosis, WILLIAM DOCK, M.D., New York, N. Y. (by invitation).
3. The First Infection Lesion in Pulmonary Tuberculosis in the Adult, HENRY ZWERLING, M.D., Berkeley, Calif. (by invitation).
4. The Roentgen Findings in Early Coccidioidomycosis, HORACE JAMISON, M.D., AND RAY CARTER, M.D., Los Angeles, Calif.

5. Pulmonary Sarcoidosis; The Early Roentgen Findings, L. H. GARLAND, M.D., San Francisco, Calif.
   Discussion: LEO RIGLER, M.D., Minneapolis, Minn., W. E. CHAMBERLAIN, M.D., Philadelphia, Penna., AND GEORGE M. WYATT, M.D., Washington, D. C.
6. Gout, EDWARD S. ROSENBERG, M.D., AND ROBERT A. ARENS, M.D., Chicago, Ill.
   Discussion.
7. The Diaphragm: A Radiologic Study in Three Dimensions, SAMUEL BROWN, M.D., Cincinnati, Ohio.

THERAPY SESSION: 2:00 P.M.

1. X-Ray Treatment of Sinusitis in Children, DONALD R. LAING, M.D., Pasadena, Calif.
   Discussion.
2. Influence on Radiation Effects Produced by Pharmacological Means, FRIEDRICH ELLINGER, M.D., Brooklyn, N. Y.
   Discussion: A. L. L. BELL, M.D., Brooklyn, N. Y.
3. Treatment of Epitheliomas of the Skin, THEODORE P. EBERHARD, M.D., Philadelphia, Penna.
   Discussion: JUAN A. DEL REGATO, M.D., Columbia, Mo.

SYMPOSIUM ON ROENTGEN DIAGNOSIS AND THERAPY OF BURSITIS AND ARTHRITIS

Chairman: E. A. Pohle, M.D.
Madison, Wis.

4. The Pathological Aspect, D. M. ANGEVINE, M.D., Madison, Wis.
5. The Roentgen Diagnostic Aspect, L. W. PAUL, M.D., Madison, Wis.
6. The Roentgen Therapeutic Aspect, E. A. POHLE, M.D., AND J. A. NORTON, M.D., Madison, Wis.

Kaufmann-Fabry Photo

Museum of Science and Industry, Jackson Park, Chicago, with Lake Michigan in the Background.

RADIOLOGICAL SOCIETY OF NORTH AMERICA

The President's invitation to the Annual Meeting of the Radiological Society of North America and the program, appearing elsewhere in this issue, make unnecessary any detailed announcement in this place. Attention is simply called to the date, Dec. 2–6, 1946, and the place, the Palmer House, Chicago. The outline of the Refresher Courses appeared in the September issue of RADIOLOGY.

UNIVERSITY OF UTAH RADIOLOGICAL CONFERENCE

Announcement has been received of the establishment of the University of Utah Radiological Conference to be held the first and third Thursdays of each month from September to June, inclusive, from 7:30 to 10 P.M., at the Salt Lake County Hospital. The conference is under the direction of the Department of Radiology of the University of Utah School of Medicine for the combined purpose of graduate instruction and staff discussion of diagnostic problems. All physicians are invited to attend and to bring with them interesting or problem cases for presentation.

The speaker at the initial conference was Dr. John Caffey, Associate Professor of Pediatrics, College of Physicians and Surgeons, Columbia University, New York.

ROCKY MT. RADIOLOGICAL SOCIETY

At the midsummer conference of the Rocky Mt. Radiological Society, held in Denver, Aug. 8–15, the following officers were elected for the ensuing year: Dr. Lewis G. Allen, President; Dr. James P. Kerby, President-elect; Dr. Ira Lockwood, First Vice President; Dr. H. M. Berg, Second Vice President; Dr. John Bouslog, Historian; and Dr. Alfred M. Popma, Secretary.

SECOND INTER-AMERICAN CONGRESS OF RADIOLOGY

Nearly three hundred reservations have been made for North American radiologists who will attend the Second Inter-American Congress of Radiology to be held in Havana, Nov. 17 to 22, according to an announcement by Dr. James T. Case, chairman of the General Committee for the United States. The block of rooms reserved at the Nacional, the headquarters hotel, have long since been disposed of. Additional reservations are being made at the Sevilla-Biltmore. Requests for hotel accommodations should be directed to Mr. Mac F. Cahal, secretary of the committee, in care of the American College of Radiology, 20 N. Wacker Drive, Chicago 6.

Delegates must arrange for air or rail transportation through their local travel agents. Special trains will be operated by the Illinois Central from Chicago and the Atlantic Coast Line from New York. Reservations for the Illinois Central should be made with Mr. J. C. La Combe, 140 South Dearborn, Chicago 3, and for the Atlantic Coast Line with Mr. R. S. Voigt, 16 East 44th Street, New York City 17.

Reservations for special flights to be operated from Chicago and New York to Miami by Eastern Airlines may be made at 120 S. Michigan, Chicago 3, or at Park Avenue and 42nd St., New York 17.

All delegates must go *via* air from Miami to Havana, and return, as no steamship service will be available. Reservation forms for Pan American Airways may be obtained from Mr. Cahal. Passports are not required.

In addition to the sixteen papers to be read by United States radiologists, there will be eighteen scientific exhibits by delegates from this country. It is expected that copies of the printed program will soon be available for registered delegates.

Representatives on the General Committee for the United States from the three cooperating societies are Dr. James T. Case, Dr. W. Edward Chamberlain, Dr. Ross Golden, Dr. Leon J. Menville, Dr. E. P. Pendergrass, Dr. B. H. Orndoff, and Mr. Mac F. Cahal.

Books Received

Books received are acknowledged under this heading, and such notice may be regarded as recognition of the courtesy of the sender. Reviews will be published in the interest of our readers and as space permits.

LE FIBRO-MYOME UTÉRIN. STATISTIQUES CRITIQUES ET COMMENTAIRES SUR UNE SÉRIE PERSONNELLE DE 1,300 CAS DONT 547 INÉDITS. FIBROMATOSE EXPÉRIMENTALE PHYSIOLOGIE DE L'OVAIRE IRRADIÉ. By J. DUCUING, Professeur de clinique chirurgicale à la Faculté de Médecine de Toulouse, Directeur du Centre Anticancéreux. A volume of 541 pages, with 156 illustrations. Published by Masson & Cie, Paris, 1946.

Books Reviewed

CLINICIAL RADIOLOGY, A CORRELATION OF CLINICAL AND ROENTGENOLOGICAL FINDINGS. Edited by CAPTAIN GEORGE UTLEY PILLMORE, MC (S), USNR, Chief Radiologist to the United States Naval Hospital, Philadelphia, during World War II; former Past Assistant Surgeon, United States Navy, during World War I; Chief Radiologist to the United States Naval Hospital, Charleston, S. C., to the United States Naval Hospital, Naval Operating Base, Norfolk, Va., to the United States Naval Hospital, Philadelphia, and to the United States Hospital Ship *Mercy* following World War I; former Chief Radiologist to the Bryn Mawr Hospital, Bryn Mawr, Penna., and to the Easton Hospital, Easton, Penna. In two volumes, 1,600 pages, with 2,484 illustrations. Published by F. A. Davis Co., Philadelphia, 1946. Price $45.00.

In the two large volumes constituting *Clinical Radiology*, edited by Capt. George U. Pillmore, we have one of the most ambitious works on roentgen diagnosis which has been produced in recent years. As Admiral Ross McIntire states in his brief Foreword, "Radiology is, of all the specialties, the one perhaps most nearly related to every other field of medicine." It is particularly fitting, therefore, that among the 58 contributors to the work there are 19 representing allied clinical branches of medicine and surgery. Many well known names appear in the list, insuring an authoritative presentation based in most instances upon a wide experience.

Volume I includes four main sections; 120 pages are devoted to The Cardiovascular System; 329 to The Respiratory System (exclusive of the paranasal sinuses, which are discussed along with injuries to the facial bones in Volume II); 243 to The Gastro-Intestinal System (more properly the digestive system, since diseases of the salivary glands, esophagus, biliary tract, and pancreas are included); 120 pages to Urology, Gynecology, and Obstetrics.

Volume II is divided into ten parts, though a number of these, all dealing with the skeletal system, might have been logically combined under a single heading. Part I (94 pages) is devoted to The Brain and Related Osseous Structures; Part II (150 pages) to The Vertebrae and Spinal Cord; Part III (54 pages) to The Nasal Accessory Sinuses and Facial Bones; Parts IV to VII, inclusive (a total of 333 pages), to lesions of the bones, joints, and cartilages, including traumatic lesions, inflammatory conditions, disturbances of nutrition and growth, and endocrine, toxic, metabolic, and static disorders. Chapters on Blood Diseases and Bone Tumors go to make up Part VIII (36 pages); The Soft Tissues, Part IX (14 pages); Foreign Body Localization, Part X (43 pages).

It is impossible in any text to include every aspect of radiology, with its many ramifications, and a few

subjects, such as roentgen examination of the teeth, have been intentionally omitted from this treatise. There is included, however, a wealth of material which will make these volumes invaluable to the radiologist and all others who are interested in this specialty.

The book is printed on excellent paper, with abundant illustrative material, beautifully reproduced. While most of the roentgenograms appear in the familiar negative form, a few of the writers have unfortunately not adhered to this plan. A useful feature is the inclusion in each volume of a complete index to the entire work. Captain Pillmore, his contributors, and the publishers are to be congratulated on this splendid publication.

X-RAYS IN PRACTICE. By WAYNE T. SPROULL, PH.D., Physicist, Research Laboratories Division, General Motors Corporation. A volume of 615 pages, including bibliography and index, with numerous illustrations. Published by McGraw-Hill Book Company, Inc., New York, 1946. Price $6.00.

Dr. W. T. Sproull, having been a research physicist in the General Motors Laboratories for more than twelve years, during which time he conducted a great deal of original research, and having had considerable experience as a teacher, both during this time and previously at the University of Wisconsin, is pre-eminently qualified for the task of writing a practical textbook on the general subject of x-rays. He has done just this in *X-Rays in Practice*.

The book is well integrated throughout. Ideas that are presented in connection with one part of the subject material are not compartmented therein to the confusion of the student, but reference is made to the application of these same ideas to other parts, so that the subject matter is a uniform and logical entity throughout.

For presentation, the subject matter is divided up along more or less classical lines, the first chapters having to do with history and the general nature of radiation. A very good chapter on x-ray-generating equipment is followed by a short chapter giving a bare outline of the concepts of radioactivity. Methods of detecting, measuring, and utilizing these radiations are presented with great clarity and a considerable amount of detail in subsequent chapters. Excellent chapters on radiography of all sorts, both for hard and soft radiation, give quite complete coverage to the subject. The largest single portion of the book, approximately 230 pages (nine chapters), is devoted to diffraction in all of its phases and applications. A short chapter is also included on electron diffraction. The broad general outlines of medical radiology are presented and there is a satisfactory chapter on protection.

Each chapter is supplemented by a section of questions and problems which are remarkably well chosen and suited to teaching purposes. There is also an appendix of useful data and a particularly clear index. The book has an excellent balance of basic theory and practical application throughout, so that at no time is the beginning student led astray or carried too far afield by either one.

In general, the text is suitable to anyone wishing a fundamental knowledge of the subject and useful for teaching in the senior or first year graduate levels. Though manufactured under wartime restrictions, the book is printed on good paper, in clear type, and is well bound.

DIE UROGRAPHIE BEI DER NIERENTUBERKULOSE. RÖNTGENDIAGNOSTISCHE UNTERSUCHUNGEN. By OLLE OLSSON. Supplementum XLVII to Acta Radiologica. A volume of 162 pages, with 50 illustrations. Published by Håkan Ohlsson Boktryckeri, Lund, 1943.

This monograph concerns renal tuberculosis leading to ulceration, caseation, and cavity formation. Other renal lesions like miliary tuberculosis and the chronic fibrosing and disseminated nodular forms are excluded. Since it is impractical to cover the contents adequately in a review, a full abstract of the treatise has been included in the Abstract Section of this issue of RADIOLOGY (p. 428).

This excellent work is profusely and beautifully illustrated, and the important value of meticulous attention to technical and diagnostic detail is obvious from its perusal. It may well change the concept of many a radiologist and clinician regarding the value of excretory urography in the diagnosis of renal tuberculosis so long as he should be willing to follow the author and his preceptor, Dr. H. Hellmer, in their excellent working manner. Urography—excretion pyelography—is of real value only if employed with the utmost attention to every detail of preparation, technic and evaluation. It will be hard to surpass the work of Olsson.

RADIOLOGICAL SOCIETIES OF NORTH AMERICA

Editor's Note.—Will secretaries of societies please cooperate by sending information to Howard P. Doub, M.D., Editor, Henry Ford Hospital, Detroit 2, Mich.

UNITED STATES

Radiological Society of North America.—Secretary, D. S. Childs, M.D., 607 Medical Arts Bldg., Syracuse 2, N.Y.

American Roentgen Ray Society.—Secretary, Harold Dabney Kerr, M.D., Iowa City, Iowa.

American College of Radiology.—Secretary, Mac F. Cahal, 20 N. Wacker Dr., Chicago 6, Ill.

Section on Radiology, American Medical Association.—Secretary, U. V. Portmann, M.D., Cleveland Clinic, Cleveland 6, Ohio.

ARKANSAS

Arkansas Radiological Society.—Secretary, Fred Hames, M.D., Pine Bluff. Meets every three months and annually at meeting of State Medical Society.

CALIFORNIA

California Medical Association, Section on Radiology.—Secretary, D. R. MacColl, M.D., 2007 Wilshire Blvd., Los Angeles 5.

Los Angeles County Medical Association, Radiological Section.—Secretary, Roy W. Johnson, M.D., 1407 South Hope St., Los Angeles. Meets second Wednesday of each month at County Society Building.

Pacific Roentgen Society.—Secretary, L. Henry Garland, M.D., 450 Sutter St., San Francisco 8. Meets annually with California Medical Association.

San Diego Roentgen Society.—Secretary, R. F. Niehaus, M.D., 1831 Fourth Ave., San Diego, Calif. Meets first Wednesday of each month.

San Francisco Radiological Society.—Secretary, Joseph Levitin, M.D., 516 Sutter St., San Francisco 2. Meets monthly on the third Thursday at 7:45 P.M., first six months of the year in Lane Hall, Stanford University Hospital, and second six months in Toland Hall, University of California Hospital.

COLORADO

Denver Radiological Club.—Secretary, Washington C. Huyler, M.D., Mercy Hospital, Denver 6. Meets third Friday of each month, Colorado School of Medicine.

CONNECTICUT

Connecticut State Medical Society, Section on Radiology.—Secretary, Max Climan, M.D., 242 Trumbull St., Hartford 3. Meetings bimonthly, second Thursday.

FLORIDA

Florida Radiological Society.—Secretary-Treasurer, Maxey Dell, Jr., M.D., 333 West Main St., S., Gainesville.

GEORGIA

Georgia Radiological Society.—Secretary-Treasurer, James J. Clark, M.D., 478 Peachtree St., N. E., Atlanta 3. Meets in November and at the annual meeting of State Medical Association.

ILLINOIS

Chicago Roentgen Society.—Secretary, Fay H. Squire, M.D., 1753 W. Congress St., Chicago 12. Meets at the Palmer House, second Thursday of October, November, January, February, March, and April.

Illinois Radiological Society.—Secretary-Treasurer, William DeHollander, M.D., St. Johns' Hospital, Springfield. Meetings quarterly by announcement.

Illinois State Medical Society, Section on Radiology.—Secretary, Frank S. Hussey, M.D., 250 East Superior St., Chicago 11.

INDIANA

The Indiana Roentgen Society.—Secretary-Treasurer, J. A. Campbell, M.D., Indiana University Hospitals, Indianapolis 7. Annual meeting in May.

IOWA

The Iowa X-ray Club.—Secretary, Arthur W. Erskine, M.D., 326 Higley Building, Cedar Rapids. Meets during annual session of Iowa State Medical Society.

KENTUCKY

Kentucky Radiological Society.—Secretary-Treasurer, Sydney E. Johnson, M.D., 101 W. Chestnut St., Louisville.

LOUISIANA

Louisiana Radiological Society.—Secretary-Treasurer, Johnson R. Anderson, M.D., North Louisiana Sanitarium, Shreveport. Meets annually at same time as State Medical Society.

Orleans Parish Radiological Society.—Secretary, Joseph V. Schlosser, M.D., Charity Hospital of Louisiana, New Orleans 13. Meets first Tuesday of each month.

Shreveport Radiological Club.—Secretary, Oscar O. Jones, M.D., 2622 Greenwood Road. Meets monthly September to May, third Wednesday, 7:30 P.M.

MARYLAND

Baltimore City Medical Society, Radiological Section.—Secretary, Charles N. Davidson, M.D., 101 West Read St., Baltimore 1.

MICHIGAN

Detroit X-ray and Radium Society.—Secretary-Treasurer, E. R. Witwer, M.D., Harper Hospital, Detroit 1. Meetings first Thursday of each month from October to May, at Wayne County Medical Society club rooms.

MINNESOTA

Minnesota Radiological Society.—Secretary, A. T. Stenstrom, M.D., Minneapolis General Hospital, Minneapolis 26. Meetings quarterly.

MISSOURI

Radiological Society of Greater Kansas City.—Secretary, John W. Walker, M.D., 306 E. 12th St., Kansas City, Mo. Meetings last Friday of each month.

St. Louis Society of Radiologists.—Secretary, Edwin C. Ernst, M.D., 100 Beaumont Medical Bldg. Meets on fourth Wednesday of each month, October to May.

NEBRASKA

Nebraska Radiological Society.—Secretary-Treasurer, Donald H. Breit, M.D., University of Nebraska Hospital, Omaha 5. Meetings third Wednesday of each month at 6 P.M. in either Omaha or Lincoln.

NEW ENGLAND

New England Roentgen Ray Society.—Secretary-Treasurer, George Levene, M.D., Massachusetts Memorial

Hospitals, Boston, Mass. Meets monthly on third Friday at Boston Medical Library.

NEW HAMPSHIRE

New Hampshire Roentgen Society.—Secretary-Treasurer, Richard C. Batt, M.D., St. Louis Hospital, Berlin.

NEW JERSEY

Radiological Society of New Jersey.—Secretary, W. H. Seward, M.D., Orange Memorial Hospital, Orange. Meetings at Atlantic City at time of State Medical Society and midwinter in Newark as called.

NEW YORK

Associated Radiologists of New York, Inc.—Secretary, William J. Francis, M.D., East Rockaway, L. I.

Brooklyn Roentgen Ray Society.—Secretary-Treasurer, Abraham H. Levy, M.D., 1354 Carroll St., Bklyn. 13. Meets fourth Tuesday of every month, October to April.

Buffalo Radiological Society.—Secretary-Treasurer, Mario C. Gian, M.D., 610 Niagara St., Buffalo 1. Meetings second Monday evening each month, October to May, inclusive.

Central New York Roentgen Society.—Secretary-Treasurer, Carlton F. Potter, M.D., 425 Waverly Ave., Syracuse 10. Meetings in January, May, and October.

Long Island Radiological Society.—Secretary, Marcus Wiener, M.D., 1430 48th St., Brooklyn 19. Meetings fourth Thursday evening each month at Kings County Medical Bldg.

New York Roentgen Society.—Secretary, Wm. Snow, M.D., 941 Park Ave., New York 28.

Rochester Roentgen-Ray Society.—Secretary, Murray P. George, M.D., 260 Crittenden Blvd., Rochester 7. Meets at Strong Memorial Hospital, third Monday, September through May.

NORTH CAROLINA

Radiological Society of North Carolina.—Secretary-Treasurer, James E. Hemphill, M.D., Professional Bldg., Charlotte 2. Meets n May and October.

NORTH DAKOTA

North Dakota Radiological Society.—Secretary, Charles Heilman, M.D., 1338 Second St., N., Fargo.

OHIO

Ohio Radiological Society.—Secretary, Henry Snow, M.D., 1061 Reibold Bldg., Dayton 2. Next meeting at annual meeting of the Ohio State Medical Association.

Central Ohio Radiological Society.—Secretary, Hugh A. Baldwin, 347 E. State St., Columbus.

Cleveland Radiological Society.—Secretary-Treasurer, Carroll C. Dundon, M.D., 11311 Shaker Blvd., Cleveland 4. Meetings at 6:30 P.M. on fourth Monday of each month from October to April, inclusive.

Radiological Society of the Academy of Medicine (Cincinnati Roentgenologists).—*Secretary-Treasurer,* Samuel Brown, M.D., 707 Race St., Cincinnati 2. Meetings held third Tuesday of each month.

PENNSYLVANIA

Pennsylvania Radiological Society.—Secretary-Treasurer, L. E. Wurster, M.D., 416 Pine St., Williamsport 8.

Philadelphia Roentgen Ray Society.—Secretary, Calvin L. Stewart, M.D., Jefferson Hospital, Philadelphia 7. Meets first Thursday of each month at 8:00 P.M., from October to May in Thomson Hall, 21 S. 22d St.

Pittsburgh Roentgen Society.—Secretary-Treasurer, Lester M. J. Freedman, M.D., 415 Highland Bldg., Pittsburgh 6. Meets second Wednesday of each month at 6:30 P.M., October to May, inclusive.

ROCKY MOUNTAIN STATES

Rocky Mountain Radiological Society.—Secretary, A. M. Popma, M.D., 220 N. First St., Boise, Idaho.

SOUTH CAROLINA

South Carolina X-ray Society.—Secretary-Treasurer, Robert B. Taft, M.D., 103 Rutledge Ave., Charleston 16

TENNESSEE

Memphis Roentgen Club.—Meetings second Tuesday of each month at University Center.

Tennessee Radiological Society.—Secretary-Treasurer, J. Marsh Frére, M.D., 707 Walnut St., Chattanooga. Meets annually with State Medical Society in April.

TEXAS

Dallas-Fort Worth Roentgen Study Club.—Secretary, X. R. Hyde, M.D., Medical Arts Bldg., Fort Worth 2. Meetings on third Monday of each month, in Dallas in the odd months and in Fort Worth in the even months.

Texas Radiological Society.—Secretary-Treasurer, R. P. O'Bannon, M.D., 650 Fifth Ave., Fort Worth 4.

UTAH

Utah State Radiological Society.—Secretary-Treasurer, M. Lowry Allen, M.D., Judge Bldg., Salt Lake City 1. Meets third Wednesday, January, March, May, September, November.

VIRGINIA

Virginia Radiological Society.—Secretary, E. Latan Flanagan, M.D., 215 Medical Arts Bldg., Richmond 19.

WASHINGTON

Washington State Radiological Society.—Secretary-Treasurer, Thomas Carlile, M.D., 1115 Terry Ave., Seattle. Meetings fourth Monday of each month, October through May, at College Club, Seattle.

WISCONSIN

Milwaukee Roentgen Ray Society.—Secretary-Treasurer, C. A. H. Fortier, M.D., 231 W. Wisconsin Ave., Milwaukee 3. Meets monthly on second Monday at the University Club.

Radiological Section of the Wisconsin State Medical Society.—Secretary, S. R. Beatty, M.D., 185 Hazel St., Oshkosh. Two-day meeting in May and one day at annual meeting of State Medical Society in September.

University of Wisconsin Radiological Conference.—Meets first and third Thursdays 4 to 5 P.M., September to May, inclusive, Room 301, Service Memorial Institute, 426 N. Charter St., Madison 6.

CANADA

Canadian Association of Radiologists.—Honorary Secretary-Treasurer, J. W. McKay, M.D., 1620 Cedar Ave., Montreal.

La Société Canadienne-Française d'Electrologie et de Radiologie Médicales.—General Secretary, Origéne Dufresne, M.D., Institut du Radium, Montreal. Meets on third Saturday of each month.

CUBA

Sociedad de Radiología y Fisioterapia de Cuba.—Offices in Hospital Mercedes, Havana. Meets monthly.

ABSTRACTS OF CURRENT LITERATURE

ROENTGEN DIAGNOSIS

The Head and Neck

WOLF, GEORGE A., JR., GOODELL, HELEN, AND WOLFF, HAROLD G. Prognosis of Subarachnoid Hemorrhage and Its Relation to Long Term Management 419

PISETSKY, JOSEPH E. Pituitary Adenoma Associated with Chronic Duodenal Ulcer..... 419

The Chest

FOSTER-CARTER, A. F., AND HOYLE, CLIFFORD. The Segments of the Lungs. A Commentary on Their Investigation and Morbid Radiology 419

BROWN, SAMUEL. The Lateral Projection of the Chest..... 420

GARLAND, L. HENRY. Intrathoracic Metallic Foreign Bodies..... 420

DE ABREU, MANOEL. The Bearers of Shadows.. 421

Tuberculosis Control in Hospitals. A Study Made by the Committee on Hospital Personnel, American Trudeau Society..... 421

BOGEN, EMIL, AND STRICKLAND, G. H. Tuberculosis in a Tropical Naval Hospital..... 421

DAYMAN, HOWARD. Silicosis: A Clinical Study. 421

RITTER, WAYNE L., AND BOVARD, PAUL G. Silicious Exposures in the Fire Brick Industry. II. Roentgenologic Study..... 422

ABBOTT, OSLER A., AND RENAULT DE OLIVEIRA, H. Spontaneous Pneumothoraces Occurring in Patients Undergoing Peroral Endoscopy.. 422

VAN ORDSTRAND, H. S., HUGHES, ROBERT, DE NARDI, J. M., AND CARMODY, MORRIS G. Beryllium Poisoning..... 422

LEACH, JOHN E. Pneumothorax in Young Adults. Descriptive Statistics in One Hundred and Twenty-Six Cases..... 422

PAUL, LESTER W. Neurogenic Tumors at the Pulmonary Apex..... 423

TAYLOR, HENRY K. Clinical Value of Angiocardiography..... 423

SOKOLOW, MAURICE, AND GARLAND, LEO H. Cardiovascular Disturbances in Tsutsugamushi Disease..... 423

The Digestive System

POPPEL, MAXWELL H., AND ROACH, JOHN F. Roentgen Diagnosis of Deformities of the Cardiac End of the Stomach, Intrinsic and Extrinsic in Origin..... 423

FULLER, CLARENCE C., AND WOOD, HAROLD. Actinomycotic Granuloma of the Stomach.. 423

TWISS, JOHN R., AND PARSONNET, EUGENE V. Practical Aspects of Peptic Ulcer Management Under Service Conditions..... 424

HODES, PHILIP J., AND KEEFER, GEORGE P. Hookworm Disease. A Small Intestinal Study..... 424

The Musculoskeletal System

MØLLER, P. FLEMMING. The Roentgen-Picture of the Tabetic Arthropathies and Affections of Bones..... 424

BLATTNER, RUSSELL J., AND SUNG, CHIEH. Cystic Tuberculosis of the Bone in a Case of Miliary Tuberculosis..... 425

SOULE, ARTHUR B., JR. Neurogenic Ossifying Fibromyopathies. A Preliminary Report.... 425

HATCHER, C. HOWARD. Pathogenesis of Localized Fibrous Lesions in the Metaphyses of Long Bones..... 426

STUBENBORD, WILLIAM D. Hereditary Deforming Chondrodysplasis..... 426

KJELLAND, P. M. A Rare Anomaly in the Elbow—Patella Cubiti..... 427

SCHNEIDER, WILMOT F. Arachnodactyly—Unusual Complication Following Skull Injury.. 427

BROWN, M. HUNTER, AND POWELL, LESTER D. Anterior Sacral Meningocele. Case Report. 427

ROBIN, PERCIVAL A., AND COLLINS, VINCENT J. Roentgenological Study of the Male Sacrum as an Aid in Caudal Analgesia..... 427

The Spinal Canal

TARLOV, I. M. Pantopaque Meningitis Disclosed at Operation..... 427

Gynecology and Obstetrics

MASON, LYMAN W. Skiodan as a Contrast Medium in Utero-Salpingography..... 427

The Genito-Urinary System

OLSSON, OLLE. Urography in Renal Tuberculosis. Roentgen Diagnostic Investigations..... 428

LEVANT, BENJAMIN, AND LEE, JAMES J. Intramuscular Urography..... 429

ESKELUND, V. Necrosis of the Renal Papillae Following Retrograde Pyelography..... 429

COOK, EDWARD N., AND KEATING, F. RAYMOND, JR. Renal Calculi Associated with Hyperparathyroidism..... 429

The Blood Vessels

RADNER, STIG. An Attempt at the Roentgenologic Visualization of Coronary Blood Vessels in Man 430
BAUER, GUNNAR. Observations on the Technique of Phlebography 430

RADIOTHERAPY

Malignant Neoplasms

ENGELSTAD, ROLF BULL. On Radio-Surgery of Tumor Metastases in the Lymph Glands of the Neck 430
WEYDE, ROLF. Treatment of Tumors of the Urinary Bladder 431
DESJARDINS, ARTHUR U. Salient Factors in the Treatment of Hodgkin's Disease and Lymphosarcoma with Roentgen Rays 431
DESJARDINS, ARTHUR U. Roentgen Treatment for Hodgkin's Disease and Lymphosarcoma of the Chest 431
ANSPACH, BROOKE M. A Review of the Problem of Cancer of the Cervix Since the Use of Radium in 1912 432
CHERNOFF, HYMAN M., EVANS, THEODORE S., AND BARTLETT, CHARLES J. Extragenital Chorionepithelioma in the Male 432

ROENTGEN DIAGNOSIS

THE HEAD AND NECK

Prognosis of Subarachnoid Hemorrhage and Its Relation to Long Term Management. George A. Wolf, Jr., Helen Goodell, and Harold G. Wolff. J. A. M. A. **129:** 715–718, Nov. 10, 1945.

The authors review the literature and consider 46 additional cases of subarachnoid hemorrhage. Twenty-nine per cent of patients entering hospitals with that condition die during the first episode of bleeding. Fourteen per cent die during recurrence between the second and fourth weeks, and another 5 per cent die by the end of the first year following the initial hemorrhage. The majority of patients surviving for a year are alive three to four years later.

Subarachnoid hemorrhage constitutes 7 per cent of all cerebral vascular disease and accounts for 2 per cent of sudden deaths. Rupture of an intracranial arterial aneurysm is most often responsible.

Symptoms and signs include sudden violent headache, dizziness, vertigo, vomiting, drowsiness, stupor, coma, stiff neck, pain in back of thighs and legs, sweats, chills, fever, Kernig sign, third nerve palsy, delirium, hemiparesis, other evidence of corticospinal tract disease, and fundal hemorrhages.

It is recommended that patients observed within four weeks after the initial hemorrhage be studied by skull roentgenograms and cerebral arteriography and that craniotomy be done if an aneurysm is visualized and if the general condition does not contraindicate surgery. Patients having had their last hemorrhage more than four weeks before initial observation, who present localizing signs of an intracranial lesion, should also have cerebral arteriography with subsequent craniotomy if indications for such are present. If a patient has survived without recurrence for more than four weeks and is without signs or symptoms, arteriography and surgical intervention should be considered optional.

H. D. Welsh, M.D.
(University of Michigan)

Pituitary Adenoma Associated with Chronic Duodenal Ulcer. Joseph E. Pisetsky. J. Nerv. & Ment. Dis. **102:** 537–546, December 1945.

Cushing (Surg., Gynec. & Obst. **55:** 1, 1932) established a substantial basis for a causal functional interrelationship between the brain and peptic ulcer. Evidently, definite centers exist in the hypothalamus which control and modulate the impulses transmitted to the gastro-intestinal tract. A disturbance in this regulatory mechanism produces tissue changes in the gastric mucosa through various intermediary steps. It seems to be irrelevant whether the disturbed cerebral function is initiated by a cerebral neoplasm, electrical stimulation, or by repeated psychic trauma. The end-result is a derailment of the physiologic control of the gastro-intestinal tract. Impulses may flow from the cortex to the hypothalamus, where they are relayed in turn to the red nucleus and then on their downward path to the centers in the medulla. There a recircuiting occurs with stimulation of the vagal and sympathetic systems, with the stomach and intestines acting as effector end-organs. The functions involved are secretion, motility, and vasomotor innervation, which are also concerned with the genesis of peptic ulcer.

The author found in the literature many specific references to peptic ulcer associated with intracranial operations or intracranial tumors. Several cases accompanying cerebellar tumors have been recorded, but relatively few have been described in association with a hypophyseal or parahypophyseal tumor. Seven cases of pituitary or parasellar lesions accompanied by peptic ulceration of the gastro-intestinal tract were found. Only one of the ulcers was in the duodenum.

The author's own patient was a 42-year-old man who had suffered from headache and pain in both eyes for fifteen years and failing vision for eighteen years. X-ray studies of the skull showed the sella turcica to be enlarged and distorted in shape. [Unfortunately the detail in the reproduction of the skull roentgenogram is too poor to show the changes described.] Physical examination revealed the expected manifestations of pituitary dysfunction. The external genitalia and testes were small, and hair growth was scant and female in distribution. The skin was pale and clear, with poor tissue turgor. No abnormal deposits of fat were present. Teleradium therapy was given, 32,500 mg. hr. and 33,000 mg. hr. to the right and left lateral temporofrontal regions, respectively. Vision in the left eye was never recovered but the patient retained enough vision in his right eye to maintain a daily routine under custodial care and was alive after a history extending over a period of twenty-four years.

The patient had given an earlier history of some abdominal symptoms, and five years after irradiation of the pituitary tumor, pain, vomiting, and nausea led to examination of the gastro-intestinal tract. The first examination showed only pylorospasm, while the second, the following year, demonstrated a deformity of the duodenal cap, giving the impression of an old duodenal ulcer. Symptoms subsequently recurred, but were relieved by a medical regimen.

Sydney F. Thomas, M.D.

THE CHEST

The Segments of the Lungs. A Commentary on Their Investigation and Morbid Radiology. A. F. Foster-Carter and Clifford Hoyle. Dis. of Chest **11:** 511–564, November-December 1945.

A bronchopulmonary segment may be defined as a segment of lung served by a principal branch of a lobar bronchus, and this branch may be called a segmental bronchus. It is usually large and fairly constant. Each branch of the bronchial tree aerates a separate wedge of lung tissue. Bronchial subdivisions usually follow a characteristic pattern and each lobe is divided into a given number of major segments.

The anatomy of the bronchial tree has been studied by casts, dissections, bronchograms, and air distention of single bronchopulmonary segments. The authors filled bronchopulmonary segments with plasticene and subsequently radiographed the specimens. The article contains illustrations showing the anatomical locations and radiological features of consolidation and collapse of all the bronchopulmonary segments.

Morbid changes of many kinds may be confined to a bronchopulmonary segment, and segmental lesions can

be detected radiographically. Identification of segmental lesions makes for more accurate diagnosis, hence for better treatment, as in bronchiogenic carcinoma, lung abscess, bronchiectasis, etc.

The authors describe the radiographic picture of each of the major segments of both lungs under conditions of consolidation and collapse. The location of the segments when *consolidated* is as follows:

A. Right Upper Lobe: (1) The *anterolateral* segment, as seen in the postero-anterior projection, extends from the hilum to the periphery, from the lesser fissure to below the clavicle. In the lateral projection, it extends from the anterior surface to the mid-axillary region, and from the lesser fissure to the first rib. (2) The *posterolateral* segment is similar to the anterolateral in location in the postero-anterior view except that the lower margin is less distinct and that usually there is a clear area between the inner border and the mediastinum. In the lateral projection it is seen posteriorly, being limited by the main interlobar fissure; it extends anteriorly to the mid-axillary region. The lower limit is on a level with the lesser fissure, and the upper portion is on a level with D2 or D3. (3) The *apical* segment occupies the apical portion of the lung. In the lateral projection it is V-shaped and extends from the hilum to the apex.

B. Right Middle Lobe: (1) In the postero-anterior projection, the *anterior* segment extends from about the fourth costal cartilage to the diaphragm, and from the right border of the heart about half way out across the lung field. In the lateral projection, it is situated anteriorly, is triangular in shape, with its apex at the hilum, limited above by the lesser fissure, laterally and inferiorly by the main fissure. (2) The *lateral* segment is seen in the postero-anterior projection as a triangular shadow in the right middle zone, with its apex down and the lesser fissure forming its base. The apex is above the diaphragm and in the mid-lung field. In the lateral projection the shadow is also triangular, extending downward and forward from the hilum, bounded above and below by the lesser and main fissures.

C. Left Upper Lobe: (1) The *anterolateral* segment is essentially the same as on the right side. The lower border is not so sharply defined, because there is no limiting fissure, and in the lateral projection the segment extends more posteriorly. (2) The *apicoposterior* segment is equivalent to the combined apical and posterolateral segments of the right lung. (The lingula, the equivalent of the right middle lobe, is supplied by the left middle bronchus, which divides into two and supplies an anterior and a lateral segment.) (3) The *anterior middle* segment in the postero-anterior projection is triangular in shape and extends from the cardiac apex laterally into the lung field in the region of the anterior ends of the 6th and 7th ribs. In the lateral projection it is triangular and extends from the hilum downward and forward and the fissure forms its inferoposterior border. (4) The *lateral middle* segment appears in the postero-anterior projection as a triangular area in the mid-zone, with its base laterally and its apex at the hilum. The medial border is superimposed on the cardiac silhouette. In the lateral projection it is triangular in shape with its apex at the hilum. Inferoposteriorly it is limited by the fissure. Its superior margin is directed downward and anteriorly. The anterior portion of the segment is superimposed over the cardiac silhouette.

D. The Lower Lobes: The two lower lobes are similar, except that the right has an additional segment, the cardiac. (1) The *dorsal* segment, as seen in the postero-anterior projection, extends across the lung field from the 5th or 6th to the 9th or 10th rib posteriorly. In the lateral projection it is triangular in shape, extending from the main fissure to the posterior chest wall, with its inferior margin sloping downward and backward towards D10; the apex may extend anteriorly to the anterior or mid-axillary region. (2) The *posterior basic* segment occupies the lower inner and posterior portion of the lung field and is triangular in shape in both projections. (3) The *anterior basic* segment extends from the hilum downward and outward and includes the costophrenic area. In the lateral projection it appears as a wedge limited anteriorly by the main fissure. (4) The *middle basic* segment, in the postero-anterior view, resembles but is more lateral than the anterior basic. In the lateral projection it is posterior to the anterior basic, based on the diaphragm. (5) The *right cardiac* segment, as seen in the postero-anterior view, occupies a small area in the right cardiophrenic region. In the lateral projection it casts a small triangular shadow based on the posterior portion of the diaphragm, with the apex pointing upward toward the hilum.

Atelectatic bronchopulmonary segments are considerably smaller and can be recognized.

HENRY K. TAYLOR, M.D.

The Lateral Projection of the Chest. Samuel Brown. Dis. of Chest **11:** 596–623, November-December 1945.

From an extensive experience, the author concludes that a roentgenographic examination of the chest made with a postero-anterior and a lateral projection yields considerably more information than a postero-anterior stereoroentgenographic study. From the lateral view, one obtains a more accurate knowledge of the size, shape, and position of abnormal shadows than is possible by stereoscopy in one direction alone, and a more accurate diagnosis is possible. Postero-anterior and lateral roentgenograms of 21 cases are included.

HENRY K. TAYLOR, M.D.

Intrathoracic Metallic Foreign Bodies. L. Henry Garland. Dis. of Chest **11:** 662–677, November-December 1945.

This report is based on a study of 150 men with intrathoracic metallic foreign bodies retained from one to twelve months following injury. The metallic fragments varied in size from less than 1 to 40 mm. in length and from 1 to 30 mm. in diameter. In approximately 20 per cent of the cases the foreign bodies measured 1 cm. or more in diameter. About 20 per cent of the patients had associated bony injuries; 75 per cent showed residual pleural changes; 15 per cent pulmonary changes; about 10 per cent showed no significant residual changes in either lungs or pleura. Included in the group were a few mediastinal and pericardial foreign bodies, one intracardiac, and a few interosseous.

The diagnosis is based on the history, presence of a scar, clinical findings, and the roentgen demonstration of a foreign body. Examination should be made in at least two planes, preferably with over-exposure, with other procedures as may be required, as tangential spot films, fluoroscopy, stereoroentgenography, tomography, and localizing procedures.

The author is especially interested in the late behavior of such cases, particularly since many survivors of the late war are hosts to these foreign bodies. In 80 per cent of the cases there are no symptoms; in 10 per cent, the symptoms may be classified as slight, and in the other 10 per cent as fairly severe. Late complications may result from migration and disintegration, erosion of blood vessels, recurrence of infection, development of herniae, pleural thickening and irritation, and pressure upon the phrenic nerve.

The author concludes that metallic foreign bodies, especially in the late stages (three or four months after injury), are frequently innocuous; that the necessity for removal does not depend upon size; that a bone fragment driven into the lung at the time of the injury is a much greater potential source of trouble than the foreign body itself. HENRY K. TAYLOR, M.D.

The Bearers of Shadows. Manoel de Abreu. Dis. of Chest **11:** 639–647, November-December 1945.

The author discusses fluorography, localized tomography, and pulmonary lavage as methods for recognizing and diagnosing tuberculosis in the "bearers of shadows." Photofluorography was considered by him as a possibility as early as 1918; he constructed his first microradiographic apparatus in 1935, and in 1937 inaugurated the first Survey Center in the Rio de Janeiro Public Health Department, using 35-mm. film. Tomographic examination is limited to shadow-bearing areas of the lung, and small films are used. Generally, 3 sections are made, at 7, 9, and 11 cm. levels from the posterior surface of the thorax. The character of the lesion can be evaluated by this method. In the ill, with little or no expectoration, pulmonary lavage is performed. This is accomplished by anesthetizing the supraglottic and infraglottic regions and instilling 10 to 20 c.c. of physiologic salt solution into the trachea during inhalation. Cough is produced, and the saline-washed material from the tracheobroncho-alveolar structure is expectorated, collected, and examined for tubercle bacilli. Bacteriological studies, cultures, and guinea-pig inoculations are also made when necessary. HENRY K. TAYLOR, M.D.

Tuberculosis Control in Hospitals. A Study Made by the Committee on Hospital Personnel (Leopold Brahdy, Chairman), American Trudeau Society. Am. Rev. Tuberc. **52:** 539–555, December 1945.

In order to be in a position to outline policies for the post-war development of the country's hospitals, the Committee on Hospital Personnel of the American Trudeau Society, with the co-operation of the American Hospital Association, sent questionnaires to 1,284 hospitals. The purpose was to ascertain which methods were being followed in order to control tuberculosis among patients and personnel. The results from 934 answers are summarized in a series of nineteen tables. About two-thirds of these hospitals tuberculin-test their student nurses while 85 per cent examine this group by x-ray. A greater proportion of the larger hospitals take periodic chest roentgenograms of their employees than of the smaller ones. Only 6 per cent x-ray the chests of all patients on admission, and only 3 per cent who do not follow this procedure plan to do so after the war emergency. This indicates the need for an educational campaign among hospital administrators and boards of directors. L. W. PAUL, M.D.

Tuberculosis in a Tropical Naval Hospital. Emil Bogen and G. H. Strickland. Am. Rev. Tuberc. **52:** 490–494, December 1945.

Tuberculosis still remains a serious source of disease, death, and disability in naval personnel despite efforts to prevent it by excluding affected recruits. In one hospital in the Southwest Pacific more than 50 patients with active tuberculosis had been seen in the past five months. In addition there were more than twice that number in whom tuberculosis was suspected but could not be proved with the time and facilities at hand.

Most of the cases developed long after the patients had entered naval service, less than a third having been in service for under two years. The roentgen findings were usually more extensive than was indicated by the history and physical findings. The majority of the patients showed widespread ill defined soft densities in the upper and middle lung fields. Infiltration and exudation were common, while fibrosis and destructive lesions with cavitation were infrequent. Few of the patients showed calcified lesions in the parenchyma or hila. Serial films, when available, showed a marked lability of the lesions.

Although the hospital from which this report was made was located close to the equator, it is not believed that the climate had any effect on the incidence or type of tuberculosis encountered. Only a few cases were found in the natives of the region and less than 10 per cent of those tested had positive tuberculin reactions. Many of the patients apparently contracted the disease while on the high seas or while stationed elsewhere, as indicated by the clinical histories. Of greater importance than the climate are thought to be the crowding together of personnel in inadequate living spaces on board ship, lack of sanitary provisions and ventilation, performance of strenuous physical exertion after the onset of symptoms, and the like.

L. W. PAUL, M.D.

Silicosis: A Clinical Study. Howard Dayman. Am. Rev. Tuberc. **52:** 449–462, December 1945.

Clinical observations on 116 patients with silicosis, 33 of whom came to necropsy, are described. Fifteen patients had simple nodular silicosis. Most of these were free of symptoms and the condition was discovered on routine examination. In the absence of complicating tuberculosis, the clinical course in this form of the disease was usually benign, some cases having been observed for periods up to eleven years without material pulmonary change.

There were 15 patients with simple conglomerate silicosis. These patients exhibited in varying degree the symptoms and signs of emphysema, with dyspnea as the initial and cardinal symptom. Roentgenograms showed areas of massive fibrosis often symmetrical and at the level of the hila. In most, nodular shadows were present as well, disseminated throughout the lung fields. The shadows remained unchanged over long periods. The disease ran a protracted clinical course with gradually increasing dyspnea. Of 8 deaths in this group, 4 were due to complicating pulmonary heart disease with cardiac failure. There was no evidence of tuberculosis. The pathogenesis of this form of silicosis is not clearly understood. It is apparent that not all cases of the disease with massive lesions can be classified as "silicosis with infection." It is possible that the bacteriologic and histologic evidence of tuberculosis has been obliterated by the silicotic process.

The third group of 77 patients had silicosis complicated by tuberculosis. The symptoms, although modified by the silicosis, were predominantly those of the tuberculous disease and included, in order of frequency, cough, expectoration, dyspnea, weight loss, fever, pleural pain, and hemoptysis. The chest roentgenogram presented widespread minute shadows similar to those seen in simple nodular silicosis together with larger mottled shadows, usually in the upper portion of the lung fields. Cavities frequently developed in the apical areas. Tubercle bacilli were found in the sputum of 65 of the 77 patients. Sixty-six of this group have died, indicating that silicosis predisposes to progressive pulmonary tuberculosis.

Heart disease due to the pulmonary disease was diagnosed in 26 of the 116 cases. The demonstration of right ventricular enlargement by means of x-ray examination was important evidence, appearing prior to any definite clinical signs. Dilatation and hypertrophy of the right ventricle are attributed to obliteration of the vascular bed of the lung and hypertension in the pulmonary artery. In tuberculosis not complicated by silicosis, such cardiac disease is rarely found.

L. W. PAUL, M.D.

Silicious Exposures in the Fire Brick Industry. II. Roentgenologic Study. Wayne L. Ritter and Paul G. Bovard. J. Indust. Hyg. & Toxicol. **27:** 283, December 1945.

To determine the hazard of silicosis to which workers in the fire brick industry might be exposed, chest films were taken, on a voluntary basis, of 876 employees (87 per cent) of seven major plants in Kentucky. An occupational history of each worker was also obtained. Ninety-seven of the men in this series, with terms of employment in the fire brick industry ranging from nine to forty-seven years, showed pneumoconiosis, this term being used because the characteristic pulmonary change found was not of the type or magnitude usually accepted in compensation courts as the discrete nodular fibrosis of silicosis. The fibrosis in these cases was uniform and finely granular, seldom showing any tendency to form discrete nodules. The chest films in two cases showed conglomerate silicosis. In both instances the men had spent their entire industrial lives, thirty-two and forty-two years respectively, operating unenclosed dry pan mills.

Twenty-six cases of tuberculosis were discovered in this survey. This is twice the ratio found on mass surveys of the general population in this area. Eleven of the 26 patients were found to have the fine granular fibrosis associated with their type of employment.

Spontaneous Pneumothoraces Occurring in Patients Undergoing Peroral Endoscopy. Osler A. Abbott and H. Renault de Oliveira. J. Thoracic Surg. **14:** 453–460, December 1945.

Little if any attention has been given to pneumothorax as a bronchoscopic hazard. Five cases are reported here in which pneumothorax complicated endoscopy (4 bronchoscopy, 1 esophagoscopy). An additional case, in which death followed bronchoscopy, is also recorded because of the similarity of the roentgen picture to that of one of the other patients. In no instance was biopsy done or a foreign body removed, so that direct injury as an etiologic factor is ruled out. The mechanism of the production of spontaneous pneumothorax is not entirely clear but most commonly there is a pre-existing tuberculosis or other pulmonary lesion, as pneumonia, gangrene, bronchiectasis, abscess, empyema, tumor, cyst, or emphysema. Rupture of such subpleural lesions may occur; in the case of endoscopic manipulation it may be precipitated by a coughing paroxysm subsequent to the procedure. It is important to recognize this complication of endoscopy so that proper treatment can be instituted when necessary. More frequent fluoroscopic and roentgenographic studies following endoscopy will probably reveal many unsuspected cases, since, as in 2 of the authors' cases, there may be no clinical symptoms. Four of the reported cases occurred during a twelve-month period in a group of 537 bronchoscopies.

HAROLD O. PETERSON, M.D.

Beryllium Poisoning. H. S. Van Ordstrand, Robert Hughes, J. M. De Nardi, and Morris G. Carmody. J. A. M. A. **129:** 1084–1090, Dec. 15, 1945.

One hundred and seventy cases of beryllium poisoning are reviewed by the authors. Manifestations included dermatitis, chronic skin ulcers, and inflammatory changes in the respiratory tract, the skin and respiratory changes occurring concurrently or singly. Diffuse pneumonitis was the severest manifestation, accounting for five deaths. In this series of cases, clinical manifestations occurred only after exposure during the processing of beryl ore. Incidence and severity were proportional to the degree of exposure and chemical irritation of dusts and fumes. The specific etiology is unknown. Use of beryllium in the production of beryllium copper alloy, widely used in industry, makes recognition of this disease important, as with increasing production more individuals may be exposed.

Forty-two patients demonstrated contact dermatitis and skin ulcers. Frequently these lesions were associated with rhinitis and nasopharyngitis. Ninety patients had a chemical nasopharyngitis and/or chemical tracheobronchitis. Chief complaints were soreness in nose and throat, with associated mild epistaxis. In 38 workers chemical pneumonitis developed, with cough, substernal pain, shortness of breath, cyanosis, in most cases an abnormal taste in the mouth, weight loss, and increasing fatigue. Onset in each instance was insidious. Signs of infection were conspicuously absent. Roentgenologic changes did not usually appear until two or three weeks after the first symptoms. Changes were bilateral and diffuse, varying with the severity of the disease. In order of their appearance they were: (1) diffuse haziness of both lungs; (2) soft irregular areas of infiltration with prominent peribronchial markings; (3) absorption of soft infiltration and appearance of discrete large or small conglomerate nodules scattered throughout both lung fields, with clearing after one to four months.

Treatment is primarily avoidance of further exposure to beryllium fumes and dusts.

N. R. SHIPPEY, M.D.
(University of Michigan)

Pneumothorax in Young Adults. Descriptive Statistics in One Hundred and Twenty-Six Cases. John E. Leach. Arch. Int. Med. **76:** 264–268, November-December 1945.

The data on 126 patients with 129 episodes of spontaneous pneumothorax observed among military per-

sonnel in the Army Air Forces Training Command are presented, largely in tabular form. In no instance did the onset occur during aerial flight. The ages of the men ranged from eighteen to forty-one years. There were no immediate fatalities. In 23 patients the onset of the spontaneous pneumothorax was gradual, and in 90 sudden. The difference between those exhibiting only mild initial symptoms or none at all (63.5 per cent) and those exhibiting moderate and severe symptoms (36.5 per cent) is statistically significant. There was no association between the severity of the reported symptoms at the onset of the attack and the degree of pulmonary collapse. In 76 patients there was nothing in the past medical history that had any reasonable association with the onset of spontaneous pneumothorax. In 85 of 100 cases the attack occurred when the patient was at rest (48.6 per cent) or during mild physical activity (37.8 per cent). The pneumothorax was of the closed type in 127 instances and of the valvular type in 2. In the majority of cases (59.8 per cent), the pneumothorax was on the right side. Slightly over one half of the patients had degrees of pulmonary collapse estimated at below 33 per cent. A search for the causative factor in 107 of the attacks was fruitless. Seventeen patients were transferred or were still in the hospital at the time of the report. Most of the men recovered without complication and were retained in military service.

Neurogenic Tumors at the Pulmonary Apex. Lester W. Paul. Dis. of Chest **11:** 648–661, November-December 1945.

Of the benign tumors most likely to develop in or at the pulmonary apex, the neurofibromas and allied neurogenic tumors are the most common. In all of these the potentiality for malignant change is present.

Benign intrathoracic neurogenic tumors, as a group, have a tendency to develop to considerable size without significant symptoms until they encroach upon or displace vital structures. Often they are discovered accidentally upon chest roentgenography for some other reason. When symptoms do develop, pain and cough are common; in tumors limited to the apex, pain radiating down the arm and a Horner's syndrome, characteristic of malignant tumors in this region, are rare. The tumor appears roentgenographically as a sharply circumscribed homogeneous shadow of variable size, situated posteriorly at the summit or along the medial aspect of the apex. The inner margin tends to merge with the mediastinum. The larger growths extend below the anatomical apex and may displace the trachea and esophagus. There is no bone destruction by invasion, though pressure erosion of ribs may occur when the tumors are large.

Benign neurogenic tumors must be differentiated from maligant growths (upper sulcus), enlarged thyroids, retrosternal thyroids, neoplasms of the thyroid and thymus and of the paratracheal and anterior mediastinal lymph nodes, aneurysms, tortuous and sclerotic innominate and subclavian arteries, and tumors arising from ribs, vertebrae, clavicles, and sternum. Metastases from the kidney or testicle must also be considered.

Surgery is the therapy of choice, as the tumors are radioresistant.

Four case histories, with roentgenograms, are included; in two cases the tumors were removed.

HENRY K. TAYLOR, M.D.

Clinical Value of Angiocardiography. Henry K. Taylor. Dis. of Chest **11:** 624–638, November-December 1945.

Filling of the heart chambers and great vessels with 70 per cent diodrast permits the differentiation of vascular from non-vascular mediastinal lesions. It is also of value for the demonstration of cardiovascular anatomy; anomalies; chamber enlargement or cardiac hypertrophy or both; contracted and expanded lesions. Six case histories and roentgenograms are included to show the value of angiocardiographic studies: one of a cardiac lesion, a ventricular aneurysm; two of non-vascular mediastinal lesions, a retrosternal thyroid and a chronic lymphatic leukemia; and three of vascular lesions, an aneurysm of the innominate artery, an arteriosclerotic aneurysm of the descending aorta, and multiple aneurysms of the aortic arch.

HENRY K. TAYLOR, M.D.

Cardiovascular Disturbances in Tsutsugamushi Disease. Maurice Sokolow and Leo H. Garland. U. S. Nav. M. Bull. **45:** 1054–1068, December 1945.

In a series of 35 patients convalescent from tsutsugamushi disease having its onset three to six months previously, the predominant symptoms were fatigability, weakness, dyspnea, and palpitation on slight effort, and precordial pains. Objective signs of residual impairment of the cardiovascular system were a decrease in duration of breath holding, abnormally rapid pulse response to standing and mild exercise, minor variations in the electrocardiogram in 7 patients, roentgen evidence of cardiac enlargement in 6 patients, and slight to moderate "peaking" of the ventricular waves in 14 cardiac kymograms. It is not known whether these disturbances are to be attributed to vasomotor instability, residual rickettsial myocarditis, or cerebral medullary lesions.

THE DIGESTIVE SYSTEM

Roentgen Diagnosis of Deformities of the Cardiac End of the Stomach, Intrinsic and Extrinsic in Origin. Maxwell H. Poppel and John F. Roach. U. S. Nav. M. Bull. **45:** 1111–1118, December 1945.

The differential roentgen diagnosis of intrinsic and extrinsic conditions producing deformities of the cardiac end of the stomach is considered, with a brief discussion of the different methods of roentgenologic examination and a listing of the various conditions in each category.

Actinomycotic Granuloma of the Stomach. Clarence C. Fuller and Harold Wood. J. A. M. A. **129:** 1163–1165, Dec. 22, 1945.

A case of primary gastric actinomycosis in a 48-year-old male, a loom fixer, is reported, with complete recovery following subtotal gastrectomy. The patient had symptoms over a five-year period, consisting of heartburn and occasional eructation and vomiting after eating. Roentgen examination disclosed deformity of the pars media due to a large penetrating ulcer on the lesser curvature just below the cardia and a persistent prepyloric deformity suggesting an additional inflammatory lesion in that region. Roentgenograms and a single photomicrograph of the operative specimen are reproduced.

JOSEPH HANELIN, M.D.
(University of Michigan)

Practical Aspects of Peptic Ulcer Management Under Service Conditions. John R. Twiss and Eugene V. Parsonnet. J. A. M. A. **129:** 857–861, Nov. 24, 1945.

Of 5,379 patients entering a large Naval hospital, 238 had disease of the gastro-intestinal tract. Included in this group were 10 proved cases of gastric and 72 of duodenal ulcer. In 46 of the 82 instances the ulcer was service-incurred, with an average period of two years between enlistment and onset of disability; 36 ulcers were not service-incurred and in these a period of approximately six months elapsed between enlistment and appearance of symptoms.

The etiology of peptic ulcer is discussed and the literature reviewed. Psychogenic constitutional states, together with problems of military service and the attendant physiologic disturbances, seem to be the essential factor in the production of ulcer. Diagnosis depends on a clinical history of pain, particularly related to meals, evidence of bleeding, and positive x-ray findings of ulcer. Such positive findings were present in 79 cases.

Confinement to bed with only bathroom privileges, a diet of milk and cream every two hours progressing to a full modified type of Sippy diet, amphojel or other antacid medication, belladonna, mild sedation, and vitamin supplements constitute treatment. All patients are treated for a minimum of six weeks, with the initial period of bed rest lasting two weeks. Control of exciting emotional factors is important. Smoking is prohibited. No liberty is granted until the patient is symptom-free and able to tolerate a regular diet.

Treatment time and modifications of the routine vary according to individual response. Improvement is based on amelioration of subjective symptoms and x-ray findings on check-up examination six weeks after the original. Follow-up films in 46 cases revealed apparent healing in 20, decrease in crater size in 10, residual deformity without crater in 8, and no change in 8. Subtotal gastrectomy was deemed advisable in 4 cases.

Thirty-five patients with service-incurred and 34 with ulcer not service-incurred were released, with 13 returning to limited duty. It is the opinion of the authors that no one with a history or findings of peptic ulcer should be admitted to the Armed Forces, and early separation from service in all cases of peptic ulcer is recommended.

H. D. WELSH, M. D.
(University of Michigan)

Hookworm Disease. A Small Intestinal Study. Philip J. Hodes and George P. Keefer. Am. J. Roentgenol. **54:** 728–742, December 1945.

Serving with a General Hospital in the Province of Assam in India, the authors had an opportunity to study hookworm disease as it developed in American troops. Their report is based upon a study of 125 patients, all with positive stools. Previous studies had indicated that hookworm disease produced changes in the roentgen appearance of the small intestine, simulating the so-called "deficiency pattern."

In 80 per cent of the authors' cases, the disease was believed to be less than three months in duration. Most of the men gave a history of itching after lying in foxholes for several hours, usually developing several hours following exposure and lasting from four to seven days. A dry cough appearing one to two weeks after the "ground itch" was common. Gastro-intestinal complaints developed from six to twenty-five weeks after exposure, pain being the most prominent symptom. Anemia was not encountered except in three instances of heavy infestation.

In 60 per cent of the patients gastro-intestinal abnormalities were demonstrable on roentgen examination. The esophagus, stomach, and duodenal bulb were usually normal. The earliest abnormalities were found in the proximal jejunum, later spreading to the distal duodenum, the distal jejunum, and the entire ileum. These changes were those of "disordered function" characterized by excessive peristalsis, segmental contractions, and distortion of the mucosal pattern. Jejunal tenderness was an early sign. Increased tone was manifested by narrowing of the lumen. Mucosal distortions varied with the severity of the disease. As it progressed, the folds became increasingly thickened and distorted. In some patients, improvement in the roentgen manifestations occurred early after the institution of treatment. In others a return to normal was delayed and the changes not infrequently persisted for months after the patient was well clinically.

The various theories offered to explain the small intestinal changes in hookworm disease are discussed. It is believed that injury to the intramural nervous system by the hook-like teeth of the parasites may be a factor.

L. W. PAUL, M.D.

THE MUSCULOSKELETAL SYSTEM

The Roentgen Picture of the Tabetic Arthropathies and Affections of Bones. P. Flemming Møller. Acta radiol. **26:** 535–547, Nov. 30, 1945.

Bone and joint changes in tabes dorsalis follow the tabetic changes in the spinal cord and are roentgenologically identical with the lesions found in syringomyelia and more rarely in other diseases of the spinal cord. The typical roentgen findings are dependent upon the increased fragility of the bone and the frequency of spontaneous fractures following slight trauma. In addition, because of the anesthesia due to the disease, the fracture is relatively painless and the patient continues to use the part with resultant comminution of the fracture, soft-tissue injury, and extensive calcification around the fracture site.

Eighty per cent of tabetic bone and joint lesions occur in the lower extremities, while in syringomyelia 80 per cent occur in the upper extremities. Characteristically, the fractures and joint lesions are associated with dislocations, extensive new bone formation, bizarre calcification in the soft tissue, and much bony débris. At the hip joint, fracture occurs close to the head, giving the effect of a shearing fracture, and is sometimes associated with almost complete absorption of the head fragment. In the spine, there are bizarre dislocations with marked bony proliferation. Changes in the knee, ankle, and foot are similar. Tabetic flatfoot is frequent, with multiple involvement of the tarsals, metatarsals, and first phalanges and the respective joints, with subsequent collapse of the arch.

Roentgen diagnosis is usually not difficult. Confusion with tuberculosis or tumor may occur, however, and secondary infection may complicate the findings. Incipient tabetic arthropathies of the hip and knee joints may be mistaken for simple arthritis deformans, especially if the changes are of a chronic nature, but the absence of subchondral cystic formations in the tabetic lesions, as well as their more rapid development, are differentiating features.

ELIZABETH A. CLARK, M.D.

Cystic Tuberculosis of the Bone in a Case of Miliary Tuberculosis. Russell J. Blattner and Chieh Sung. J. Pediat. **27:** 579–582, December 1945.

Tuberculous processes in the long bones of children may be divided into two groups according to location, those in the diaphysis and those in the metaphyso-epiphyseal region. A lesion in the diaphysis is modified by periosteal reaction, while one in the metaphyso-epiphyseal region results in a cystic structure. The latter is a comparatively rare condition, especially in infants or very young children, the first case in the United States having been reported in 1931 (Schwentker: Am. J. Dis. Child. **42:** 102, 1931).

A case of cystic tuberculosis of the metaphysis and epiphysis of the tibia in a 2-year-old child, with miliary tuberculosis and tuberculous meningitis, is reported. On routine fluoroscopy, all of the bones appeared normal except for a defect at the site of the tuberosity of the right tibia. Careful palpation revealed an area of softness here. There was no swelling of the subcutaneous tissue and no sign of periosteal reaction. The knee joint was not involved. The bony defect was filled out by caseous material which, being less radiopaque, gave the lesion a cystoid appearance on the anteroposterior view and a punched-out appearance on the lateral view. There was no roentgen evidence of bony regeneration or sequestral formation. Direct smear of material (4 c.c. thick white pus admixed with blood) aspirated from the area of the bony defect revealed numerous acid-fast bacilli, many in clumps. Inoculation of this material into two guinea-pigs produced tuberculosis. The patient died on the eighth hospital day. Autopsy showed an acute miliary tuberculosis of the lungs, liver, spleen, and leptomeninges. Microscopic section of a fragment of necrotic bone removed from the right tibia revealed typical tuberculous involvement.

Neurogenic Ossifying Fibromyopathies. A Preliminary Report. Arthur B. Soule, Jr. J. Neurosurg. **2:** 485–497, November 1945.

Sixty-one patients who had sustained trauma of the spinal cord and one with spinal cord disease of unknown nature, all with paraplegia of the lower extremities, were examined radiographically. "Scout" films of the lower extremities were made in the anteroposterior projection. Additional views—stereoscopic anteroposterior, lateral, and oblique projections—were taken of the affected parts of some of the patients. "Scout" roentgenograms were also taken of the upper extremities in several cases with abnormal findings in the lower extremities, but none of these revealed any abnormalities.

In all but 4 of the 61 cases of traumatic origin there had been immediate complete loss of motor and sensory function distal to the involved areas. At the time of examination, these 4 patients showed clinical signs of improvement, and roentgenograms revealed absence of osteoporosis and no evidence of abnormal ossifications. In 23 other traumatic cases there were on admission clinical signs of returning function. Fifteen of these patients had lesions of the cauda equina. Only 3 of the improved patients had ossifications in the soft tissues.

Of the 27 improving patients, 10 showed varying degrees of osteoporosis in the bones of the lower extremities. In the entire series of 62 patients, the only ones who had marked osteoporosis were 2 of those showing improvement, both with lesions of the cauda equina, and one unimproved patient with osteoporosis in the bones of each tarsus but not elsewhere.

Of the 35 unimproved patients, 20 showed soft-tissue ossifications—3 about both hips and both distal femora; 2 about both hips and one distal femur; 6 about both hips alone; 3 about one hip alone; 4 about both distal femora alone; 2 in one distal thigh alone. Of the 23 improved and unimproved patients with ossifications, 11 showed no osteoporosis, 8 slight osteoporosis, and 4 slight osteoporosis near the hips and knees and moderate osteoporosis in the bones about the ankles and in the feet. Of the patients with ossifications, 19 had lesions of the thoracic cord with dermatome levels ranging from T-3 to T-10; one had a lesion of the lower cervical cord with sensory level at C-7; 3 had lesions of the cauda equina with motor and sensory loss below the L-1 dermatome.

There was no significant difference in the blood total protein and calcium levels in the patients with ossifications and those without. In those with ossifications, however, the average blood phosphorus level was 4.7 mg. and the average phosphatase level was 6.2 mg., as compared with 3.9 mg. and 5.2 mg. in the absence of ossifications.

In the early stages, the soft-tissue deposits had a more or less amorphous, cloud-like roentgen appearance, similar to that observed in developing ossification of a hematoma. Even in the earliest cases, however, there was a definite tendency toward linear and trabecular structure.

In all patients examined three months or more after injury, the deposits were characterized by trabeculated cancellous bone with slender, interlocking strands enclosing small ovoid or lozenge-shaped spaces between them. While some of the masses were discrete and moderately dense, in other areas the bone was laid down in sheets or bands, which appeared to be distributed along muscle or fascial planes.

About the hips, the deposits were extracapsular and most extensive above and anterior to the femoral necks and greater trochanters, although scattered osseous masses appeared about all margins and surfaces of the hip-joint capsules. When well developed, the deposits partially ensheathed the hip, extending from the pelvis about 1 cm. peripheral to the outer margins of the acetabulum to the upper shaft of the femur, to which they were attached a short distance below the lesser trochanter. The deposits about the knee were most prominent in the region of the medial collateral ligament, where in several cases they resembled the ossifications of Pellegrini-Stieda disease. In most cases, however, they were more extensive, with bands of osseous tissue extending upward over the arch of the medial condyle, where they appeared to be attached to the periosteum of the distal third of the shaft of the femur. Ossifications were less abundant lateral to the knee, but here, also, they assumed a pattern similar to those on the medial side.

Some of the ossifications terminated abruptly at the level of the knee joint space. In other cases, they extended about 1 cm. beyond the joint space. No ossifications were observed anterior or posterior to the knee. Films in several cases showed isolated small islands of bone deep in the soft tissues of the thigh.

Soft-tissue ossifications of the extremities, chiefly in the vicinity of the hips and knees, constitute an apparently common complication of injury or disease of the spinal cord and cauda equina. This condition is to

be differentiated from the atrophic type of lesion with impaired motor nerve function and the osteoarthropathic type of lesion with impairment of afferent nerve function. Due to the preliminary nature of this study, no conclusions are drawn. Several avenues of future investigation are suggested.

Pathogenesis of Localized Fibrous Lesions in the Metaphyses of Long Bones. C. Howard Hatcher. Ann. Surg. **122:** 1016–1030, December 1945.

Localized benign fibrous lesions situated in the metaphyses of long bones have been described under a variety of diagnoses. Over-emphasis on various secondary characters prominent in the natural course of this condition accounts for the diverse interpretations placed upon it. It has been described as solitary xanthoma, solitary bone cyst, benign giant-cell tumor, non-suppurative fibrous osteomyelitis, and as non-osteogenic fibroma of bone.

The present report is based on a study of 45 patients in whom 51 lesions of the type under discussion were identified. In all cases of this group, the lesions were in the long bones of the lower extremities: 32 in the distal portion of the femur, 11 in the proximal portion of the tibia, 5 in the distal metaphysis of the tibia, and 2 in the distal third of the fibula [one unaccounted for]. All of the lesions were situated in the metaphysis or the adjacent shaft, where they are usually eccentrically placed. The defect often abuts on the cortex or occupies a portion of it. In 5 cases, multiple lesions were present. In all but 7 of the patients, the abnormality was recognized in childhood, indicating that the disorder has its inception during the period of longitudinal bone growth.

Symptoms are usually mild. In several cases, the lesion was discovered incidentally in making radiographic studies for other purposes. Pain, when present, is usually slight, intermittent, and often referred to the neighboring joint. Local tenderness may be present when the area is in, or close to, the cortex. Symptoms or physical findings of epiphyseal disturbances led to the discovery of the associated metaphyseal defect in 14 of the present series.

The roentgenographic appearance of the metaphyseal defect is distinctive and along with the clinical findings should make the diagnosis quite certain. Early in the disease, there is a limited area of reduced density in the metaphysis close to the epiphyseal disk. Later, the area is apt to be elongated in the direction of the axis of the bone and show sharp demarcation from surrounding normal bone by a shell of bony sclerosis. Irregularly scalloped margins give the appearance of loculation. Later on, through longitudinal growth of the bone, the defect comes to lie farther from the epiphyseal cartilage. In the process of tubulation of the metaphysis, the defect may eventually occupy a part of the cortex. In that event, periosteal reaction may produce the shadow of overlying periosteal new bone. In lesions long present there may be radiographic evidence of progressive ossification at the periphery, with resulting marginal sclerosis several millimeters in width. Sometimes dense ossification may fill out part or all of the area, leaving a zone of increased density in the radiograph.

In 17 cases of this group, material was removed for study. The pathological appearance varies with the age and activity of the lesion just as does the radiological appearance. Different areas in the same focus may yield a wide variety of findings. The basis of the lesion in all stages is a fibrous connective tissue which occupies a smooth-walled, sometimes partially loculated, cavity in the bone. The recently formed focus shows a relatively cellular fibrous tissue with scattered multinucleated cells while, in what appears to be a somewhat older lesion, the fibrous tissue is arranged in strands and whorls. Numerous areas of recent hemorrhage and blood pigment are seen. Multinucleated cells may be more numerous than in the early stage. Lymphoblasts are found scattered through the fibrous tissue. Lipoid-filled macrophages are often present in focal collections or diffusely scattered throughout defects where radiographic evidence indicates a lesion of long duration. The lipoid deposition is evidence of chronicity and not a primary feature of the disorder.

Disturbances in the epiphyseal bone and cartilage were found in conjunction with the metaphyseal defect in 14 of 45 patients. Osteochondritis of the tibial tubercle or Osgood-Schlatter's disease was the most typical disturbance, present in 8 patients. Osteochondritis dissecans of the femoral condyle was present in 4 cases, and osteochondritis of the patella in 2. Disturbance of growth from the epiphyseal cartilages about the knee from unknown cause was associated with a defect in the femoral metaphysis in 1 patient.

Since the metaphyseal defects are found commonly in childhood and rarely in adult life, it is obvious they must undergo spontaneous healing. The defect found in the metaphysis of a young child may heal rapidly by reparative ossification or, if it is located near the margin of the metaphysis, longitudinal growth and tubulation of the bone may cause it to be displaced to the outer surface of the bone and obliterated. If early repair does not occur, and the lesion is so situated that it is not carried to the outer surface, it usually becomes encapsulated by a dense sclerotic wall. Such a focus may then undergo gradual obliteration by ossification from its walls. Sometimes this will result in restitution of normal architecture of the region, but sometimes a localized hypertrophy of bone will remain to mark the site of the previous lesion.

The author gives several case histories illustrative of various aspects of this disease, with excellent radiographic reproductions. The etiology is still entirely unknown, but the association with demonstrable epiphyseal disorder is considered significant. The only treatment indicated is for the associated disorders.

BERNARD S. KALAYJIAN, M.D.

Hereditary Deforming Chondrodysplasis. William D. Stubenbord. South. M. J. **38:** 816–819, December 1945.

Hereditary deforming chondrodysplasia, or Ollier's disease, is a developmental disorder, the chief features of which are multiple exostoses and irregularities in growth of the epiphysis. The disease is congenital in origin and frequently several members of the same family are affected. Transmission is said to take place through the male.

The author reports a case in a girl of 13, whose paternal grandfather, mother, and several siblings were known to have similar deformities. Roentgenograms of the long bones are reproduced, showing multiple exostoses along the course of muscle attachments, arising in the metaphyseal portions of the long bones.

FRANCIS B. MARKUNAS, M.D.

A Rare Anomaly in the Elbow—Patella Cubiti. P. M. Kjelland. Acta radiol. **26:** 491–496, Nov. 30, 1945.

Roentgenographic findings in two cases of patella cubiti are presented. In one case, arthrography was done and extension of the joint cavity upward, similar to the suprapatellar bursa at the knee, was demonstrated. In both instances the anterior surfaces of the bones appeared to be covered with cartilage. In neither case was there a history of trauma sufficient to have caused fracture with subsequent pseudarthrosis.

Ontogenetically, patella cubiti occurs in several of the lower orders, and in man the epiphysis for the olecranon is sometimes double. From these observations, the author concludes that patella cubiti is a true accessory bone. ELIZABETH A. CLARK, M.D.

Arachnodactyly—Unusual Complication Following Skull Injury. Wilmot F. Schneider. J. Pediat. **27:** 583–588, December 1945.

This paper should perhaps have been entitled "Marfan's Syndrome Complicated by Skull Injury." The author traces the appearance, in his patient, of the various anomalies found in Marfan's syndrome—delayed development apparent before the age of two, a spontaneous fracture of the femur, probably due to fragility of the long bones, between the ages of two and four, and progressive loss of vision, for which no glasses were prescribed. At the age of thirteen, when almost blind, the patient fell down an open cellar door. She was unconscious for some time and received cuts about the arms, legs, and eyes. Within two to three weeks the left eye became prominent. Ophthalmologic examination revealed subluxation of the lens, retinal detachment, and bilateral glaucoma. The patient's mental age was approximately five years and the intelligence quotient 37. The typical clinical and roentgen features of Marfan's syndrome—"spider hands and feet," rarefaction and spindly appearance of the bones of the hands and feet, with decreased subcutaneous tissue—were present.

Anterior Sacral Meningocele. Case Report. M. Hunter Brown and Lester D. Powell. J. Neurosurg. **2:** 535–538, November 1945.

The clinical and roentgenographic features in a successfully treated case of anterior sacral meningocele are described. Roentgenograms of the lumbar spine and coccyx showed the characteristic "scimitar sacrum." Myelography, with pantopaque, demonstrated free communication between an extended terminal theca and the upper portion of the meningocele sac. The technical aspects of primary excision and closure are presented with a view to standardizing surgical treatment of this unusual lesion.

Roentgenological Study of the Male Sacrum as an Aid in Caudal Analgesia. Percival A. Robin and Vincent J. Collins. Anesthesiology **6:** 505–514, September 1945.

In 50 unselected cases the sacrum was studied roentgenologically prior to caudal analgesia. Subsequently the independent observations of the anesthesiologist and roentgenologist were analyzed. A routine anteroposterior view of the sacrum was obtained to determine the apex of the caudal canal and to measure the transverse diameter of the caudal hiatus at the level of the fourth sacral segment. For the lateral view, body-section roentgenography was used to delineate more accurately the caudal canal. Three lead markers were placed on the skin along the approximate mid-line of the sacrum and three or four planigraphic sections were made close to the mid-line. A routine lateral view of the sacrum employing good roentgen technic, resulting in sharp end-points, is probably equally reliable. The following points were disclosed in the lateral film: the anteroposterior diameter of the opening of the caudal canal, the caliber of the lumen of the entire canal, the size of the cornua, and the configuration of the sacral curve.

In 8 patients the needle could not be introduced into the caudal canal, and in 3 the caudal canal was entered by force. The roentgen findings in these cases are described. The significant anatomic features, revealed by roentgenography, which contributed to unsuccessful analgesia were a narrowed anteroposterior diameter, absence of hiatus, blocked lumen, and agenesis of the posterior wall of the caudal canal. The importance of roentgen studies prior to caudal analgesia is emphasized.

THE SPINAL CANAL

Pantopaque Meningitis Disclosed at Operation. I. M. Tarlov. J. A. M. A. **129:** 1014–1016 Dec. 8, 1945.

A single case of pantopaque meningitis in a 37-year-old male is reported. Myelography was done in course of investigation of chronic low back pain. Neurologic examination was negative. Five hours following intrathecal injection of 3 c.c. of pantopaque the patient complained of generalized headache and stiffness of the neck and there was temperature elevation to 100.8°. On the following day the temperature rose to 102° and a day later, when interlaminar exploration was done, nuchal rigidity persisted, although fever had lessened. No evidence of herniated disk was found. The spine and lamina of L4 were removed, and on opening the dura and arachnoid considerable whitish, soft, stringy exudate adherent to the arachnoid and nerve roots of the cauda equina was seen. Pantopaque was removed from the subarachnoid space.

Histopathologic study of the exudate disclosed a meshwork of fibrin strands with embedded polymorphonuclear leukocytes, lymphocytes, and plasma cells. No organisms were cultured from this material.

In view of the symptoms, there is little doubt that inflammatory reaction was widespread within the subarachnoid space. Recovery was uneventful.

This report serves to emphasize a potential hazard attached to the use of pantopaque. To lessen the likelihood of such reaction, immediate complete removal of the medium following myelography is indicated.

JOSEPH HANELIN, M.D.
(University of Michigan)

GYNECOLOGY AND OBSTETRICS

Skiodan as a Contrast Medium in Utero-Salpingography. Lyman W. Mason. Rocky Mountain M. J. **42:** 942–943, December 1945.

Because of the extreme slowness with which lipiodol is absorbed from the peritoneal cavity and the irritative effect of the retained oil in certain instances, the authors employed skiodan for uterosalpingography in two cases. The latter medium, which has long been

used intravenously for pyelography, proved relatively non-irritating in the peritoneal cavity and its absorption was rapid and complete. Four or 5 c.c. is believed to be enough for the average case. The visualization obtained appears from the illustrations to be clear and adequate.

The author claims no originality for the use of skiodan for uterosalpingography, stating that "it is possible that others have used it for the same purpose." [He is correct in this assumption, for Titus, Tafel *et al.* reported the use of a skiodan-acacia mixture for uterosalpingography as early as 1937 (Am. J. Obst. & Gynec. **33:** 164, 1937; Ibid. **36:** 889, 1938). In their experience, skiodan alone had not sufficient viscosity for this purpose.—Ed.] PERCY J. DELANO, M.D.

THE GENITO-URINARY SYSTEM

Urography in Renal Tuberculosis. Roentgen Diagnostic Investigations. Olle Olsson. Acta radiol. suppl. 47, pp. 1–162, 1943.

This monograph concerns renal tuberculosis leading to ulceration, caseation and cavity formation. Other renal lesions, like miliary tuberculosis and the chronic fibrosing and disseminated nodular forms, are excluded.

In the introduction it is stated that a diagnosis of renal tuberculosis usually can be made without roentgen examination. Reasons for x-ray examination are: (1) the resultant shortening of diagnostic procedure, (2) the possibility of earlier diagnosis, (3) the possibility of diagnosis of closed foci, (4) a differentiation between forms of renal tuberculosis amenable to surgical interference and other types, (5) a desire to have more than circumstantial evidence on which to base a serious surgical decision, though it is emphasized that the diagnosis of renal tuberculosis should not be based on radiologic evidence exclusively.

These questions are proposed for evaluation: (1) Are the contrasts obtained sufficient for a study of the anatomy of the renal pelves? (2) Is it permissible to draw conclusions concerning the extent of the destructive tuberculous process in the renal parenchyma from the density of the contrast shadows obtained in the renal lumina? Thus, the attempt is made to furnish an answer in regard to anatomic and functional criteria on which the diagnosis might be based. From the answer obtained, information was expected as to whether urographic changes could be anticipated in all cases of renal tuberculosis and whether such changes permitted of an etiologic diagnosis.

The study was based upon 89 patients with 173 kidneys, among them 103 tuberculous kidneys, observed between June 1, 1933, and December 1942 in the Roentgen Diagnostic Department of the University of Lund, these figures comprising all proved cases of renal tuberculosis seen at this institution in that time. The patients ranged in age from ten to sixty-one years and presented symptoms from one month to five years, though it should be mentioned that 12 were entirely symptomless. Quite a number of these patients stated that they had experienced originally a short period of illness and distress, followed by a long interval, up to several years, of well-being before more protracted recurrent symptoms led to a detailed study. One-third of all patients experienced acute colic-like episodes as their first symptom, easily misleading to a suspicion of urinary calculi.

Technical procedures were standardized as far as possible. The intestinal tract was cleared well on the day preceding the examination, by castor oil and enemata. On the day of the examination, breakfast and material amounts of fluid were omitted, while the examination was scheduled as early in the morning as possible in order to avoid undue accumulation of gas in the intestinal tract. While, in the beginning, uroselectan was used as contrast material, this was soon abandoned for perabrodil, which is now used exclusively. Excluded from the procedure were all patients with a non-protein-nitrogen level exceeding 50 mg. No serious reactions were encountered, but all questionable patients were tested for hypersensitivity by intradermal preliminary injections.

The radiologic procedure was regulated as follows: (1) A scout film of the abdomen was obtained. If shadows suspicious of calculi were observed, they were investigated further and localized by additional oblique and lateral films as necessary. (2) The second exposure was obtained 1 to 4 minutes after the intravenous injection and reviewed immediately. (3) Immediately following this exposure, gradual compression was applied to the lower ureters and after this had been in place for 5 minutes, additional films in various numbers were made as considered desirable. (4) After removal of the compression, supplementary views of ureters and bladder, often including various oblique projections, were secured. Postero-anterior projections not infrequently were obtained and found of advantage when ventrally located cavities within the kidneys were to be demonstrated.

A detailed chapter of this monograph deals with urographic diagnostic criteria of renal tuberculosis based on functional observations as follows:

About one-quarter of all cases of renal tuberculosis examined presented normal contrast shadows of the renal pelves. The density of the contrast shadows, therefore, cannot be considered exclusively and by itself as the sole criterion for or against the existence of a tuberculous process. This applies especially to early cases of limited extent.

One-seventh of all tuberculous kidneys failed to give any contrast urograms. These kidneys were involved by advanced disease processes, and 14 of the 15 revealed changes suggestive of disease in the scout films—irregular enlargement, calcific deposits, "putty kidneys—" while only one appeared grossly normal. In these cases urography proved of importance in so far as it gave information concerning the functioning kidney, which is often hard to obtain instrumentally because of severe existing bladder involvement.

Two-thirds of the cases showed varying degrees of functional impairment which was of diagnostic importance in so far as advanced cases generally showed pronounced functional deficiency, while milder degrees of dysfunction generally corresponded to smaller lesions. However, exceptions occurred in both directions, and their significance and possible causes are discussed in some detail. They detract from the value of urography, especially in consideration of bilateral lesions. They emphasize the necessity of evaluation of all available functional and anatomic data.

The next chapter analyzes urographic criteria of renal tuberculosis based on studies of anatomic detail. In order to evaluate roentgenologic possibilities in this respect, the earliest anatomic manifestations of the disease are discussed in contrast to tuberculous bacilluria

and non-surgical forms of renal tuberculosis, including the so-called nephrocirrhosis. European and American authors follow a somewhat divergent line of argumentation, but the conclusion may be drawn that "the early form of clinical renal tuberculosis represents in pathologic anatomic concepts a solitary papillary ulceration communicating with the renal pelvis." This is amenable to roentgenologic demonstration by contrast delineation, and its roentgen diagnostic symptom is the demonstration of a papillary defect; in other words, it is analogous to the "niche symptom" of a gastric ulcer.

Regarding as minimal cases only those with involvement of a single papilla, the author gives four instances of such a demonstration. It is emphasized that ureteral compression is to be applied for quite a long time in order to obtain such optimal demonstration of minimal lesions. As sources of error are mentioned: minor variations in the normal shape of calices; beginning pyelovenous and tubular reflux; non-tuberculous papillary necrosis in pyelonephritis, especially of diabetics; tumor, pyelitis cystica, caliceal diverticula, pyelogenous cyst, and blood clots. Exposures at various angles may lead to a differential diagnosis, together with a careful evaluation of all clinical data.

It should be kept in mind that a pathologic process may be of such minute size that it can escape detection; furthermore, there is hardly any chance for radiologic detection if the lesion does not communicate with the lumen of the pelvis, or if the communication is not patent to contrast fluid.

Changes in the configuration of the renal lumina result from expanding or shrinking parenchymal processes and the dislocations produced thereby. In rare instances the pelvis may be infected primarily and such involvement may lead to a caliceal stenosis which, however, will require a most careful analysis, especially with due regard to the primary anatomy of the organ and its lumina.

Reviewing his entire material, the author points out that all cases but one—102—presented urographically demonstrable changes. He emphasizes, however, that the roentgen image not infrequently may be regarded as characteristic for tuberculosis, but should not be considered as pathognomonic by itself. A diagnosis of renal tuberculosis on an exclusively radiologic basis is not justified. It should be the object of a radiologic examination either to arouse or confirm a suspicion of such infection. The radiologic indication for a nephrectomy should be a demonstration of pathologic changes rendering such surgical intervention advisable, regardless of whether or not they may be specific in nature.

The remainder of the monograph contains detailed case histories.

For a critical evaluation of this work, see the Book Review section in this issue of RADIOLOGY (page 414).

H. A. JARRE, M.D.

Intramuscular Urography. Benjamin Levant and James J. Lee. Pennsylvania M. J. **49:** 255–257, December 1945.

Investigation of the urinary tract by the intramuscular injection of diodrast is considered by the authors to be the method of choice in children. It has the advantage over the intravenous and subcutaneous methods of being easily and quickly carried out, with a resultant minimum of crying.

The patient is placed face downward on a table and a 35 per cent (undiluted) solution of diodrast is injected intramuscularly into each buttock, with a 21-gauge needle and a 20-c.c. syringe. The total dose varies from 10 to 20 c.c. A flat plate is taken before the injection, and films are taken ten, twenty, and thirty minutes later.

Tests for sensitivity may be done by placing a drop of the solution in the eye, the intradermal test, or placing 2 c.c. of diodrast in the mouth for two minutes and having the patient swallow it.

The authors recommend a cathartic, overnight dehydration, and no breakfast. If the abdomen is shown by the flat plate to be full of gas, castor oil and enemas are given and the patient returns for urography two days later.

The films obtained by the method described have been superior to those obtained by the intravenous and subcutaneous methods. Forty-two children ranging in age from one month to fifteen years have been examined. There have been no local or general reactions.

JOSEPH T. DANZER, M.D.

Necrosis of the Renal Papillae Following Retrograde Pyelography. V. Eskelund. Acta radiol. **26:** 548–554, Nov. 30, 1945.

Although retrograde pyelography is usually without complications except for mild local irritation, the possibility of damage to the kidneys of patients with poor renal function and whose general condition is unfavorable should be borne in mind. The case reported here occurred in a 55-year-old woman with a known chronic pyelonephritis which had not responded to drug therapy. The procedure was done with 25 per cent Hippodin (sodium ortho-iodine hippurate) and was followed by reduction in urinary output, rise in blood, urea nitrogen, and fever. The patient died in uremia less than one month later.

At autopsy the kidneys were rather small, with loss of normal markings on cut section and with necrosis of the apices of the papillae. Grossly, no crystalline precipitates were visible. Microscopically, there was acute and chronic pyelonephritis, with a number of small crystalline bodies within the tubules. These "microliths" had neither the characteristics of sulfathiazole nor of other crystalline bodies usually found in the kidneys. The complete necrosis was confined to the renal papillae.

Because of the immediate reaction following pyelography, the subsequent course, which lasted almost four weeks, and the microscopic findings, the author presents the case as one of renal damage due to Hippodin probably superimposed on damage due to previous sulfathiazole therapy in diseased kidneys.

ELIZABETH A. CLARK, M.D.

Renal Calculi Associated with Hyperparathyroidism. Edward N. Cook and F. Raymond Keating, Jr. J. Urol. **54:** 525–530, December 1945.

The purpose of this paper is to re-emphasize the relationship between hyperparathyroidism and renal calculi, which the authors find to be more often associated than osteitis fibrosa and parathyroid disease. Albright, who in ten years accumulated a series of 67 cases of hyperparathyroidism, found classical osteitis fibrosa cystica in only one-third and maintained that renal involvement was more frequent and its conse-

quences more important. The present writers reaffirm this. In eighteen months they collected 18 cases of proved hyperparathyroidism. In 4 of these there were associated classical bone changes alone, while 14 of the patients had renal calculi.

Symptoms of hyperparathyroidism vary widely. Constitutional symptoms include general muscular weakness, lassitude, fatigue, and constipation, ascribed to hypercalcemia. Polyuria and polydipsia occur, and the specific gravity of the urine is usually low. In the presence of renal calculi the symptoms are those commonly seen with that condition. Skeletal symptoms consist in vague aching in the bones and joints, pathological fractures, cysts and tumors of bone, and skeletal deformities.

The diagnostic criteria of hyperparathyroidism are hypercalcemia, hypophosphatemia, hypercalcinuria, and hypophosphaturia. The serum calcium and phosphorus values fluctuate, however, and one negative examination does not rule out the disease, as subsequent studies may yield positive findings. Hyperparathyroidism should be seriously considered when the serum calcium exceeds 10.5 mg. per 100 c.c. With borderline calcium levels, the finding of a low total serum protein may be indicative of hyperparathyroidism. The concentration of alkaline phosphatase in the serum is not increased except in the presence of bone lesions.

In cases of hyperparathyroidism associated with renal calculi a detailed urologic study is indicated. Roentgenologic findings may show only a single stone or there may be long standing calculous disease and diffuse or miliary calcinosis of the renal parenchyma. In the authors' series most of the stones were of calcium oxalate. The treatment is removal of the parathyroid adenoma or hypertrophied tissue, which should usually be done before removing the calculi, to avoid recurrences. Renal emergencies, however, may necessitate handling the urologic problem first.

Hyperparathyroidism should be given due consideration in all cases of renal calculi, especially if recurrent, regardless of the bone findings.

ARTHUR W. PRYDE, M.D.

THE BLOOD VESSELS

An Attempt at the Roentgenologic Visualization of Coronary Blood Vessels in Man. Stig Radner. Acta radiol. **26:** 497–502, Nov. 30, 1945.

Angiography of the cerebral vessels and of the heart and great vessels has been accomplished, but opacification of the coronary arteries presents a more difficult problem. By puncturing the bulb of the aorta it was found that the coronary arteries could be filled in animals. The author's method in human subjects is puncture of the aorta through the sternum under fluoroscopic control and injection of thorotrast. The procedure was carried out on 5 patients. Faint outlines of the proximal portions of the vessels can be seen in the roentgenograms, although the superimposed shadows of the pulmonary veins complicate interpretation. There were complications in two cases, mediastinal emphysema in one, and extravasation of blood into the pericardium and subsequent exudative pericarditis in the other. Both patients recovered without sequelae.

ELIZABETH A. CLARK, M.D.

Observations on the Technique of Phlebography. Gunnar Bauer. Acta. radiol. **26:** 577–588, Nov. 30, 1945.

Of about 500 phlebographic examinations, 180 were performed because of clinical suspicion of incipient thrombosis in the lower legs. Diodrast was the preferred contrast medium, but Intron (sodium diiodomethane sulfonic acid) was also satisfactory if diluted with physiological saline and followed by injection of saline when pain occurred. Injections were made into the vein behind the lateral malleolus after exposure through a small skin incision, and roentgenograms were taken in anteroposterior and lateral projections. All patients were examined with the leg in horizontal position.

In 73 of the 180 patients examined, no visualization of the deep veins was obtained, and the diagnosis of thrombosis was considered established. In 62 patients the veins were so well filled that they were regarded as normal, but in only 44 of these was opacification perfect. Of the remaining 18 "normal" roentgenograms, 10 showed a thin-line filling of one or more segments due to settling of the dye along the dependent surface; 6 showed small defects interpreted as blood from tributary veins, and 2 cases showed combinations of these findings. Of the 45 patients with incomplete filling, which was interpreted as incipient thrombosis, 42 subsequently gave typical clinical evidence of the condition. In 18 of these latter examinations, filling was absent in a long segment of a major trunk, with dilatation of the vein below; in the remaining 27 there were irregular bubble-like rarefactions in the dye-filled vein.

ELIZABETH A. CLARK, M.D.

RADIOTHERAPY

MALIGNANT NEOPLASMS

On Radio-Surgery of Tumor Metastases in the Lymph Glands of the Neck. Rolf Bull Engelstad. Acta radiol. **26:** 555–576, Nov. 30, 1945.

Since there is wide variation in the method of treatment of cervical lymph node metastases and since clinical diagnosis of metastasis is uncertain, the author presents a series treated at the Norwegian Radium Hospital from 1923 to 1944 and advocates the combination of block dissection and radiation therapy in selected cases. Among malignant tumors which metastasize to the cervical nodes, carcinoma of the lip and oral cavity are the most important; cancers of the tonsils, pharynx, larynx, salivary glands, nose, sinuses and skin produce metastases less commonly. These latter, grouped together under the heading "other tumors," usually present problems of more importance than the cervical metastases and are considered only under special circumstances for the type of therapy described.

The author bases indications for block dissection first on prognosis. Since the incidence of metastasis from carcinoma of the lip is moderate and the primary lesion can usually be controlled, the patient is kept under careful observation and block dissection is not per-

formed until suspected metastases appear and only after these have failed to respond to radiation. In young patients, however, and in those having rapidly growing metastases, surgical treatment is primary, with roentgen therapy postoperatively. In cancer of the oral cavity, the incidence of metastasis is higher and control of the primary lesion is more difficult. In these cases the adjacent nodes are always included in the irradiation of the primary tumor and when malignant involvement is suspected the entire neck region is irradiated. Block dissection is postponed until the radiation reaction has subsided. The nodes may then be dissected if there is a reasonable chance that the primary tumor is curable. If there is no reasonable chance of cure, extensive surgical intervention is contraindicated, with a few exceptions in which it may be done for palliation. In almost all cases, block dissection should be carried out only if there is a high probability that radical dissection can be accomplished.

In the series reported, a total of 113 block dissections were carried out on 104 patients, with an operative mortality of 1.8 per cent. At the time of the report, 43 patients (41.3 per cent) were alive without evidence of recurrence; 50 (48.1 per cent) had died of the cancer; 2 were alive with cancer, and 9 had succumbed to intercurrent disease (a new primary cancer in 4 instances). The author bases his survival rates on the number of patients followed. Thus, of 23 patients with carcinoma of the lip observed for three years, 19 or 82.6 per cent were symptom-free at the end of that period, while of 20 followed for five years, 16 or 80 per cent were without evidence of disease. For cancer of the oral cavity, the survival figures (symptom-free) are 38.5 per cent of 39 patients after three years and 33.3 per cent of 30 patients after five years. In the miscellaneous group of "other tumors," the three-year figure is 35.7 per cent of 14 patients and the five-year figure 37.5 per cent of 8 patients. Taking the group as a whole, 51.3 per cent of 39 patients followed for three years were alive and well, and 50 per cent of 29 patients followed for five years.

Of the entire 104 patients, only 14.4 per cent had recurrences at the site of dissection. Histologic diagnosis confirmed the clinical impression of metastasis in 79.7 per cent of 118 cases (including some in which only partial dissection was done), but in the remaining 20.3 per cent no metastases were found.

The anatomical relationships of the cervical lymph nodes are discussed and the surgical procedure for block dissection is described. ELIZABETH A. CLARK, M.D.

Treatment of Tumors of the Urinary Bladder. Rolf Weyde. Acta radiol. **26:** 589–605, Nov. 30, 1945.

The author's choice of method in the treatment of bladder tumors is electrocoagulation and implantation of radium needles in the base of the tumor followed by roentgen irradiation. He recommends cystotomy for the coagulation and implantation of radium because the tumor can be evaluated more successfully and implantation can be accomplished with greater accuracy. Modifications of the ideal plan of therapy become necessary when the poor general condition of the patient and the extent of the tumor make surgery impractical: thinness of the bladder wall may contraindicate implantation of radium needles, and the tolerance for radiation may not allow administration of the dose of 5,500 r considered desirable. In young people with papillomas and in small recurrences electrocoagulation through the cystoscope, if the patient can be kept under observation, is considered satisfactory. Resection was possible in a few of the author's cases, and electrocoagulation after response of previously inoperable tumors to radiation in a few others. Tumors which showed atypical cells in otherwise characteristic papillomas in the older age groups were treated as carcinomas because of the tendency to recurrence with frank malignant changes found early in the series.

Of 155 patients with tumors of the urinary bladder, 53 were in such poor condition that not even palliative radiation could be administered. At the time of compilation of statistics, a total of 111 patients had died of their bladder neoplasms and 4 of intercurrent diseases. Of the 39 living patients, 28 were free from recurrence; 11 patients were living free from symptoms for more than five years, and 20 for more than three years. In only 13 patients could the combined method of surgical-radiological treatment, with the insertion of radium needles, be carried out, and this group is considered too small for a reliable expression of results. ELIZABETH A. CLARK, M.D.

Salient Factors in the Treatment of Hodgkin's Disease and Lymphosarcoma with Roentgen Rays. Arthur U. Desjardins. Am. J. Roentgenol. **54:** 707–722, December 1945.

The treatment of Hodgkin's disease and lymphosarcoma is covered in considerable detail and some aspects of clinical recognition and course of the diseases are discussed. It is pointed out that roentgen therapy may be of use in differential diagnosis, especially when the lesions are confined to the mediastinum. In determining the plan of treatment, the areas of maximum involvement should receive first attention. The author prefers to use roentgen rays generated at moderate voltages (130 to 140 kv.) and filtered through 4 to 6 mm. of aluminum. He believes this quality of radiation is more effective than that produced by higher kilovoltages even for the treatment of mediastinal or abdominal node involvement unless the patient is above the average in size. He has found it more effective to give each field treated, in one or two days, as large a dose of radiation as can be tolerated without harmful effects. This dose usually is between 550 and 600 r (surface dose, measured in air). Different fields are treated successively, one at a time, until all have received the specified dose. The time distribution of treatment is of considerable importance. Intervals between treatment to any single area of as long as a week and the use of too small a dose at each individual treatment are major factors in poor regression of the lesions and the development of early radio-resistance.
L. W. PAUL, M.D.

Roentgen Treatment for Hodgkin's Disease and Lymphosarcoma of the Chest. Arthur U. Desjardins. Dis. of Chest. **11:** 565–589, November-December 1945.

Comment is made on the specific sensitivity of different types of cells. The changes occurring in sensitive cells, such as lymphocytes, after irradiation, are described. The sensitiveness of tumors corresponds closely to that of the cells of which the tumors are chiefly composed. Knowledge of comparative sensitiveness of tissue permits of differential diagnosis following a therapeutic test. Thus a clear distinction can be made between Hodgkin's disease or lymphosarcoma and

an aortic aneurysm, carcinoma of a bronchus, neurofibroma, fibrosarcoma, teratoma, or a tuberculous adenitis.

Hodgkin's disease and lymphosarcoma, clinically, are essentially the same. They are members of the same family. They can start, develop, and invade from and to the same places in precisely the same manner and at the same rate. They are also influenced by a given dose of x-rays in about the same manner and at the same rate. In most cases where mediastinal nodes are invaded, the involvement is secondary to lymph node disease in other regions. When axillary nodes are invaded, mediastinal nodes are usually involved. Also, when inguinal nodes are invaded, the retro-abdominal nodes are involved.

Hodgkin's disease and lymphosarcoma are composed largely of hyperplastic lymphoid cells and are very sensitive to roentgen rays. In the majority of cases, an intrathoracic lymphoblastomatous mass can be expected to shrink 30 to 100 per cent within three to four weeks after a well planned course of treatment. The author treats through two large anterior and two corresponding posterior fields. Each anterior port extends from the suprasternal notch to the ensiform cartilage, and from the mid-line to the anterior axillary line. The central rays converge through each port at an angle of 30 to 40°, being directed to the central part of the chest. Treatment is also given through two additional fields, directed to the mediastinum through the thoracic inlet, including the neck, if nodes in the upper part of the chest are involved. When retro-abdominal nodes are involved, additional treatment is directed toward the upper half of the abdomen through two anterior and two posterior fields, or toward the entire abdomen through four anterior and four posterior fields.

The author has observed better results with intermediate than with deep therapy. He prefers 130 or 140 kv. with 6 mm. Al filtration and 550 r to each field in one or two days, to 200 kv. and 600 r to each field in three to six days. He feels it is wise to repeat the same dosage in three weeks, even if there appears to be 100 per cent regression of the involved nodes, unless there is a marked reduction in the white count. The regression and improvement last longer after two courses of therapy. It may be necessary to give three courses; then the interval between the second and third courses should be lengthened to five or six weeks.

HENRY K. TAYLOR, M.D.

A Review of the Problem of Cancer of the Cervix Since the Use of Radium in 1912. Brooke M. Anspach. Am. J. Obst. & Gynec. **50:** 681–690, December 1945.

In 1911 Wertheim was obtaining an 18.4 per cent five-year absolute survival rate following operation for the removal of cancer of the cervix in patients in whom his percentage of operability was 50. Kelly and Burnam began an epoch in 1912 by adopting radium as the treatment of choice. Ever since, gamma rays have been a large factor in the treatment of cancer and to a considerable extent have taken the place of radical surgery.

Anspach discusses the present means of finding the early cases of cervical cancer and the danger of promiscuous use of hormones near the menopause, which may prolong the "delay period" before the diagnosis is established. The color test of Schiller, colposcopy, and vaginal smear methods direct attention to suspected cases, but biopsy or diagnostic curettage remains the trustworthy criterion.

In irradiation the lesion must be studied as to exact position and extent. "The acme of radium application is achieved only when a sufficient supply of radium salt or its emanation suitably screened is at hand at the moment of application." The dose is 3,600 mg. hr. and upwards. A "massive dose" at the first treatment has been the trend for a long while. Heyman and others believe in smaller repeated applications. Alpha and beta rays are excluded by platinum capsule walls of 1.5 mm. thickness or 2.8 mm. of lead, when the content is 50 mg. of radium salt. For 10 mg. needles, 0.5-mm. platinum or 0.03-mm. solid gold walls are satisfactory.

Theoretically, deep x-ray therapy should precede radium application, since manipulation incident to radium application may displace cancer cells not previously exposed to deep x-ray. The standard treatment used by the author consists of 1,600–2,400 r given through four ports. A transvaginal port exposure of the same dose has been added since 1944.

Bonney succeeds Wertheim as the outstanding exponent of radical surgery and in 1941 reported his absolute five-year salvage in 500 operations as 25 or 26 per cent.

Lynch, comparing irradiation and an advanced radical operation in a selected group with irradiation alone, favored definitely the combination of irradiation and operation. For example, in the ten-year estimate, the salvage rate of the combination was 62.9 per cent, and of irradiation alone, 36.7 per cent.

In summary the author states that a greater number of cancer patients are presenting themselves in an early stage; that irradiation will become more widely and adequately available, and more effectual; that operative risk has been reduced and improvement is likely to continue; that a combination of irradiation and operation will perhaps give better results.

RUSSELL WIGH, M.D.

Extragenital Chorionepithelioma in the Male. Hyman M. Chernoff, Theodore S. Evans, and Charles J. Bartlett. Arch. Int. Med. **76:** 347–351, November-December 1945.

A case of extragenital chorionepithelioma in a 30-year-old male is recorded. The clinical picture of gynecomastia, pain in the lumbar region, hemoptysis, loss of weight, and rapid downhill course in this patient is characteristic of tumors of embryonal urogenital origin which have metastasized widely. Intensive roentgen therapy over a period of five months did not alter the outcome, although it did produce some symptomatic relief and some decrease in the size of the pulmonary metastases.

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